Exhibit 1

UNITED STATES DISTRICT COURT
DISTRICT OF NEW JERSEY

IN RE: JOHNSON & JOHNSON TALCUM POWDER PRODUCTS MARKETING, SALES PRACTICES, AND PRODUCTS LIABILITY LITIGATION ***THIS DOCUMENT RELATES TO ALL CASES***	**Civil Action No. 3:16-md-2738-FLW-LHG** **MDL No. 2738**

AFFIDAVIT OF JACK SIEMIATYCKI, MSc, PHD

I, Jack Siemiatycki, do hereby declare as follows:

1. I am over the age of 18 years of age and otherwise competent to testify to the matters contained in this affidavit.

2. I have been retained by the Plaintiffs in this case as an expert witness.

3. I have reviewed Defendants' Reply in Support of their Motion to Exclude Plaintiffs' Experts' General Causation Opinions, which cites to and draw inferences from a recently published article that I contributed to entitled "Shift Work Patterns, Chronotype, and Epithelial Ovarian Cancer Risk". Leung, et al., *Shift Work Patterns, Chronotype, and Epithelial Ovarian Cancer Risk*, Cancer Epidemiol Biomarkers Prev 2019;28:987-995, attached as Exhibit A. There were nine co-authors; I was neither the lead author (Leung) nor the senior author (Koushik).

4. Defendants misinterpret a sentence from this report and incorrectly refer to it as evidence that my opinions differ between the courtroom and the greater scientific community.

5. The following are my responses to these and related inferences drawn by Defendants in this article. The fact that I do not address any other points made by Defendants in their motions to exclude my testimony and opinions should not be interpreted as an expression of agreement with such point(s). All statements of fact are true and correct, and all opinions are based on, amongst other things, on my training, experience, and research.

6. In light of the foregoing, the following are my responses:

Overview of My Opinions and Misinterpretation by Defendants.

7. In their reply brief regarding general causation, Defendants refer to a single sentence in a recent article I contributed to as evidence that my opinions in this litigation are limited to the courtroom, and that I espouse different opinions in the greater scientific community.

8. As quoted below, the specific sentence in the Leung et al article that Defendants refer to and emphasize lists risk factors for ovarian cancer, but does not include talc:

> "While the etiology is not well understood, established and strongly suspected risk factors include older age, never use/short duration of use of oral contraceptives, low parity, personal history of breast cancer, family history of breast or ovarian cancer, use of hormone replacement therapy, increased height, and a high body mass index (BMI; refs. 2, 3)."

Defendants suggest that I specifically chose to not include talc use as a risk factor for ovarian cancer in this sentence because, outside of the litigation context, I do not believe that talcum powder products are a risk factor for or cause ovarian cancer. *See* Defendants Johnson & Johnson and Johnson & Johnson Consumer Inc.'s Reply in Support of Motion to Exclude Plaintiffs' Experts' General Causation Opinions at 3, 73-74, 81-82.

9. To be clear and unequivocal, my opinion both in this litigation and in the general scientific community is that talcum powder product use causes and is a risk factor for the development of ovarian cancer. These opinions are explained in great detail in my previously submitted expert witness report. *See* Rule 26 Expert Report of Jack Siemiatycki MSc, PhD, dated November 16, 2018, attached as Exhibit B.

10. The sentence in question here from the Leung et al article was not included in any of the drafts that I reviewed prior to publication. Speaking to Dr. Koushik, I have since learned that the sentence was added to the article shortly before publication at the insistence of an anonymous reviewer. This specific addition, and other minor revisions made to the article which were insisted upon by the anonymous reviewer, were not circulated by the senior author for my review or for review by any other co-authors prior to publication. It is common practice in multi-author scientific publications for the senior author to exercise discretionary privilege to decide when

an issue that arises in response to reviewers' comments requires a multilateral consultation with co-authors.

11. Whatever the intentions of the anonymous reviewer for insisting on insertion of this sentence, it is important to keep in mind that this sentence appears in an article wholly unrelated to talc and that the sentence clearly reads as a non-exhaustive list of risk factors.

12. A reasonable scientist would not read the non-inclusion of talc here as an affirmation that talc is not a risk factor for ovarian cancer.

Brief Chronology of Publication on Shift Work and Ovarian Cancer.

13. In 2008, under the leadership of Dr. Koushik, a team including myself devised a research protocol to investigate the causes of ovarian cancer. Among the many exposures that the team proposed to investigate in relation to ovarian cancer were hormonal factors, physical inactivity, and shift work. The latter factor was the ultimate focus of the article Defendants refer to on shift work and ovarian cancer. This article is one of several that have been published based upon our data collection, and several additional articles are currently being prepared for future publication.

14. Over the course of several years, the team collected data, completed large portions of scientific data analyses, and began work to publish individual articles on individual exposure associations with ovarian cancer.

15. In 2017, Dr. Koushik began work with a trusted graduate student Lisa Leung to produce a draft manuscript on the relationship between disrupted sleep patterns and risk of ovarian cancer.

16. In August 2018, a draft of the manuscript was circulated to the entire research team for comment. The sentence quoted by Defendants above was <u>not</u> part of the draft manuscript that was circulated to the co-authors, nor was there any passage in the draft which conveyed the concepts in the sentence quoted above. I reviewed the draft manuscript, and sent some comments and suggested revisions to Dr. Koushik.

17. In October 2018, after assessing comments made by the various colleagues who participated in this project including myself, Dr. Koushik and her student revised and submitted the manuscript to the journal Cancer Epidemiology, Biomarkers & Prevention for publication.

18. In November 2018, the journal indicated the article would be accepted for publication, subject to a small number of minor revisions requested by anonymous

reviewers. The sentence referenced by Defendants here was one of the minor revisions insisted upon by one of the anonymous reviewers.

19. As the revisions had no impact on the basic methods, results, or interpretation of data specifically applicable to shift work and ovarian cancer, Dr. Koushik saw no reason to send out those suggestions to other co-authors and accepted the suggested revisions in order to not delay the publication process.

20. The journal accepted the article with accepted revisions in December 2018 and published in March 2019.

Conclusion.

21. Defendants here refer to a single sentence not written by me and not circulated for my review from an article wholly unrelated to talcum powder products as demonstrable proof that I hold different opinions about talcum powder products and ovarian cancer outside the litigation context.

22. It is my opinion, regardless of the setting in which I express it, that talcum powder products cause and are a risk factor for the development of ovarian cancer.

FURTHER AFFIANT SAYETH NOT.

JACK SIEMIATYCKI, MSc, PH.D.

SWORN AND SUBSCRIBED TO before me this 3rd day of July 2019:

Notary

Exhibit A

Research Article

Cancer Epidemiology, Biomarkers & Prevention

Shift Work Patterns, Chronotype, and Epithelial Ovarian Cancer Risk

Lisa Leung[1,2], Anne Grundy[1,3], Jack Siemiatycki[1,3], Jocelyne Arseneau[4], Lucy Gilbert[4], Walter H. Gotlieb[5], Diane M. Provencher[1,6], Kristan J. Aronson[2,7], and Anita Koushik[1,3]

Abstract

Background: Shift work causing circadian disruption is classified as a "probable carcinogen" and may contribute to the pathogenesis of hormone-sensitive cancers. This study investigated shift work exposure in relation to epithelial ovarian cancer (EOC) risk.

Methods: In a population-based case–control study with 496 EOC cases and 906 controls, lifetime occupational histories were collected and used to calculate cumulative years of shift work exposure, average number of night shifts per month, and average number of consecutive night shifts per month. ORs and 95% confidence intervals (CI) for associations with EOC risk were estimated using logistic regression. Associations were also examined according to chronotype and menopausal status.

Results: More than half of the cases (53.4%) and controls (51.7%) worked evening and/or night shifts. There was no clear pattern of increasing EOC risk with increasing years of shift work; the adjusted OR of EOC comparing the highest shift work category versus never working shift work was 1.20 (95% CI, 0.89–1.63). This association was more pronounced among those self-identified as having a "morning" chronotype (OR, 1.64; 95% CI, 1.01–2.65). Associations did not greatly differ by menopausal status.

Conclusions: These results do not strongly demonstrate a relationship between shift work and EOC risk.

Impact: This study collected detailed shift work information and examined shift work patterns according to shift times and schedules. The findings highlight that chronotype should be considered in studies of shift work as an exposure.

Introduction

Ovarian cancer is a deadly disease, ranking as the fifth leading cause of cancer-related death among women in Canada and the United States (1). While the etiology is not well understood, established and strongly suspected risk factors include older age, never use/short duration of use of oral contraceptives, low parity, personal history of breast cancer, family history of breast or ovarian cancer, use of hormone replacement therapy, increased height, and a high body mass index (BMI; refs. 2, 3). Shift work causing circadian rhythm disruption was classified as a "probable carcinogen" by the International Agency for Research on Cancer (IARC) in 2007 (4). In several epidemiologic studies, long-term shift work has been associated with increased cancer risk at multiple sites (5), with the majority of research focused on breast cancer (6–8). The dominant mechanistic focus has been on the "melatonin hypothesis," which postulates that exposure to light at night interferes with circadian rhythms by suppressing melatonin production (9–12) and elevating circulating levels of estrogen, and that if this hormone disruption occurs over many years, the risks of breast and endometrial cancers are increased (13–15). Strong experimental evidence has supported this mechanism and has suggested that this pathway may extend to other hormone-sensitive malignancies, such as epithelial ovarian cancer (EOC; refs. 16, 17).

Four epidemiologic studies have previously assessed shift work exposure in relation to EOC risk (18), two reporting a positive association (19, 20), and two observing no evidence of an association (21, 22). Differences in findings may be related to differences in the sources of data and shift work definitions across these studies that included self-reported occupational history with specific questions about night work (19), current baseline rotating work (20), self-reported years of working rotating shifts with nights (21), and census-based job information linked to a job-exposure matrix indicating percentage of shift workers for a given job title (22). Also, there is substantial variability in the organization of shift work (e.g., timing of shifts, schedule of days/nights, duration of shifts, number of consecutive shifts), and there is some evidence implying that certain work patterns, such as a greater number of consecutive night shifts, may disrupt circadian

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Note: Supplementary data for this article are available at Cancer Epidemiology, Biomarkers & Prevention Online (http://cebp.aacrjournals.org/).

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rhythms more than other parameters (23–25). However, epidemiologic studies have often aggregated shift work patterns differing in timings and schedule into one or two overall measurements (23).

Discrepancies between studies may also be attributed to the lack of consideration of chronotype, that is, an individual's biological preference as a "morning," "intermediate," or "evening" person (26), which is potentially an important effect modifier of the relationship between shift work and cancer. A person's circadian rhythm is synchronized to sleep and wake times through the regulation of physiologic processes such as the production of melatonin, where people with "evening" chronotypes are synchronized to evening time periods (i.e., melatonin production peaks later), and can sleep and wake later with ease, while people with "morning" chronotypes prefer the opposite (27). Research has shown that people with "evening" chronotypes may be more tolerant to shift work (28, 29), which may suggest that the mechanism by which shift work impacts cancer risk may differ across chronotypes. Chronotype has been considered in only one previous ovarian cancer study where the findings suggested an increased risk associated with shift work among those self-identified as "morning" people, with weaker relative risk estimates for "evening" people (19). Another potential effect modifier is menopausal status as supported by a recent combined analysis of breast cancer studies, where shift work was associated with an increased risk among premenopausal women only (30). EOC is a hormone-sensitive cancer and it is hypothesized that shift work may elevate estrogen levels through light at night-induced endocrine dysregulation, and this may be differential by menopausal status.

In a population-based case–control study, we investigated the relationship between shift work exposure and risk of EOC overall, by tumor behavior (invasive, borderline), and separately for high-grade serous carcinoma (HGSC), the most common form of EOC. Associations were also examined according to chronotype and menopausal status.

Materials and Methods

Study population

The PRevention of OVArian Cancer in Quebec (PROVAQ) study is a population-based case–control study conducted in Montreal, Canada in 2011–2016 (31). All study participants were women ages 18–79 years who were Canadian citizens, residents of the metropolitan area of Montreal, and able to communicate in French or English. Cases were women newly diagnosed with EOC, including primary peritoneal and fallopian tube cancers, and recruited from seven Montreal hospitals that care for the large majority of women diagnosed with ovarian cancer in Montreal. A total of 652 women with histologically confirmed EOC were eligible and asked to participate, of whom 78% ($n = 507$) gave consent to participate. Nine participants were later excluded as their cancers were nonepithelial or metastatic, leaving 498 cases. Cases were classified by tumor behavior (invasive, borderline) as well as on histology and grade (32). Population controls were identified from the Quebec Electoral List and were frequency-matched to cases on five-year age categories and electoral district. Of 1,634 eligible controls asked to participate, 56% ($n = 908$) agreed to participate. All cases and controls provided written informed consent.

Data collection

In-person interviews were used to ascertain sociodemographic information, medical history, medication use, reproductive characteristics, anthropometric measurements, other lifestyle factors, and lifetime occupational history including shift work details for each job held. On the basis of the question "Do you consider yourself to be a morning person, more morning than evening, more evening than morning, or an evening person?" participants self-reported their chronotype. Information pertinent to the determination of menopausal status (31) was also collected during the interview. Interviews were conducted an average of 4.8 months after diagnosis for cases. Occupations were classified according to the International Standard Classification of Occupations 1968, by an occupational hygienist, based on job titles and description of tasks.

Shift work assessment

For each job, volunteer activity, period as a full-time graduate student or period as a homemaker held for at least six months over the age of 19 years until the referent age (age of diagnosis for cases, age of interview for controls), participants reported the job title, duration each job was held, status (part-time, full-time), work pattern [fixed days (6 am–6 pm), fixed evenings (6 pm–12 am), fixed nights (12 am–6 am), rotating (alternating day shifts with night/evening shifts), or other], number of night shifts per month, and number of consecutive night shifts per month. For work patterns reported as "other," participants provided a short statement describing their exact schedule that was later categorized into one of the predefined questionnaire work patterns. Periods reported as a homemaker were considered a fixed day pattern. Eight participants who were students aged 25 and younger at recruitment reported no prior employment and were classified as having a fixed day work pattern from age 19 to their referent age. Two controls and two cases were excluded due to incomplete occupational history, leaving 496 cases and 906 controls for analysis.

The IARC Monographs defined shift work as "any arrangement of daily working hours other than the standard daylight hours of 7/8 am–5/6 pm" (4), which encompasses work patterns of fixed evenings, fixed nights, and rotating (with either evening shifts or night shifts). We defined three shift work exposure variables: cumulative years of shift work exposure, average number of night shifts per month, and average number of consecutive night shifts per month. Cumulative years of exposure was calculated for any shift work as well as for individual shift work patterns defined on the basis of shift times (ever night shift work, evening shift work only) and schedules (rotating shift work only, fixed shift work only; Fig. 1). The ever night shift work exposure group included participants exposed to night shifts only as well as to both evening and night shifts. We were unable to include a group restricted to participants exposed to night shifts only due to a small number of exposed participants. Cumulative years of shift work exposure were calculated as in Eq. (A):

$$C = \sum_{i=1}^{n} (D_i \quad F_i) \qquad \text{(A)}$$

where C is lifetime cumulative years of exposure, n refers to the total number of jobs with shift work across a participant's lifetime, i refers to a specific job with shift work, D is job duration in years, and F is part-time or full-time equivalency (0.5 for part-time, 1.0 for full-time). The variable for cumulative years of exposure to any shift work was categorized into tertiles based on shift working

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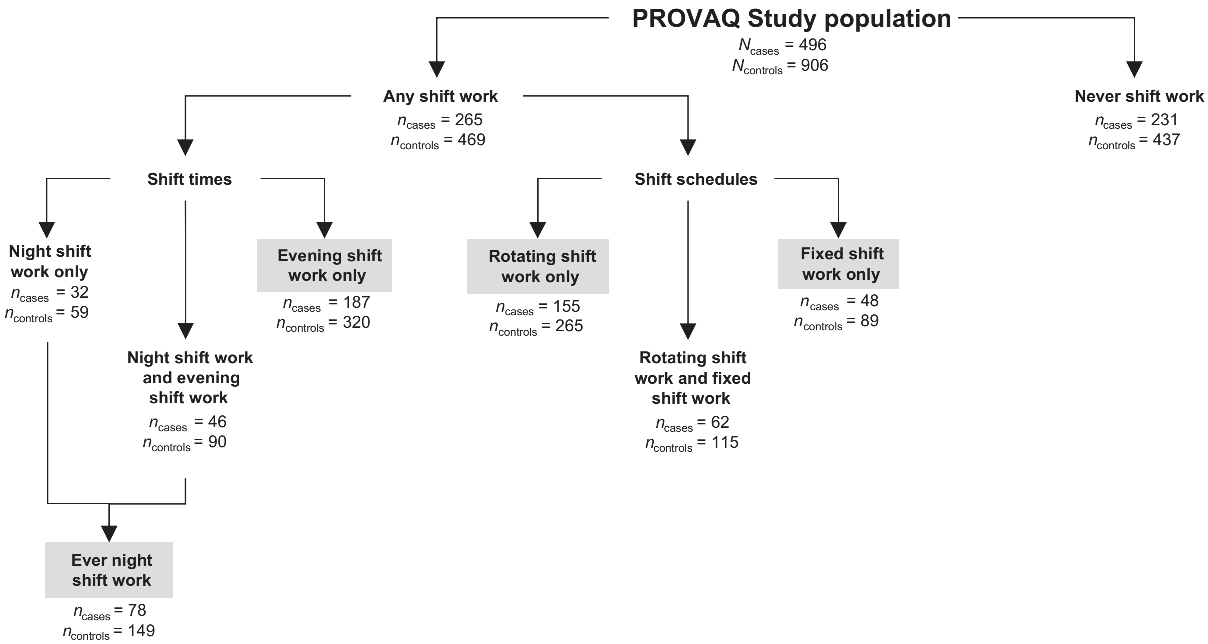

Figure 1.
Shift work exposure assessment and deconstruction of the any shift work exposure variable into individual shift work patterns (shaded) included in the analysis: ever night shift work, evening shift work only, rotating shift work only, and fixed shift work only.

controls; for individual shift work patterns, cumulative years of exposure was dichotomized on the basis of the median among shift working controls. For participants exposed to night shifts, the number of night shifts per month was determined by taking an average of the number of night shifts per month from all positions involving night shifts. For the analysis, exposure categories were created by dichotomizing at the median among ever night shift working controls. The exposure categories for the variable for average number of consecutive night shifts per month were produced using the same method. Because the question on the number of consecutive night shifts was added after the study began, ever night shift workers who were never asked the question were excluded from the analysis of these variables (15 cases, 25 controls).

Statistical analysis

Multivariable unconditional logistic regression was used to estimate ORs and 95% confidence intervals (CIs) for the associations between overall EOC risk and each shift work exposure variable, with women without shift work experience (i.e., never shift workers) as the reference group. Confounders of the shift work and EOC association were identified using directed acyclic graphs (DAG) combined with change-in-estimate procedures (33). Potential confounders that were considered are indicated in our DAG (Supplementary Fig. S1) and included age, ethnicity, family history of ovarian cancer, education level, BMI, parity, breastfeeding duration, duration of oral contraceptive use, history of tubal ligation, hormone replacement therapy use, endometriosis, medically diagnosed infertility and smoking history. From these variables, we identified a minimally sufficient confounder set, which all models were adjusted for, that included age (continuous), education (<high school, high school, college/technical, University undergraduate, University graduate), and parity (nulliparous, 1, 2, 3 full-term births). In the last step of this confounder selection method, each variable not included in the minimally sufficient confounder set was reevaluated (33); no other variable was identified as a confounder. P_{trend} across exposure categories was calculated by considering the category ranks as a continuous variable in the logistic regression model and evaluating the Wald χ^2 test statistic with one degree of freedom to test for a linear effect on the logit of the probability of EOC or EOC subgroup.

Multivariable polytomous logistic regression was used to estimate ORs and 95% CIs for the associations according to tumor behavior (i.e., invasive and borderline). Heterogeneity in the associations by tumor behavior was tested using likelihood ratio tests that compared a model where ORs were constrained to be equal among subgroups, to a model where ORs were allowed to differ between subgroups (34). We evaluated whether ORs for shift work and overall EOC risk were modified by chronotype (morning, intermediate, evening) and by menopausal status (premenopausal, postmenopausal) by including product terms for shift work and the effect modifier of interest. These analyses were conducted for cumulative years of exposure to any shift work, as well as for cumulative years of exposure to evening shift work only and rotating shift work only; sample sizes were too small for ever night shift work and fixed night shift work only. *P* values for multiplicative interaction were produced using likelihood ratio tests comparing the regression models with and without the product terms.

In three separate sensitivity analyses to examine the possible influence of reverse-causality bias and/or a lagged effect of shift work given the possible induction period of ovarian cancer, occupational history 2, 5, and 10 years prior to referent age were excluded from the cumulative years of exposure variable for cases

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and controls. Two sensitivity analyses addressing the categorization of shift work were conducted where the variable for any shift work exposure was dichotomized (ever, never) and categorized using cutoffs from studies of breast cancer (<15, 15–29, >29 years). All statistical analyses were conducted using SAS software version 9.4 (SAS Institute).

Results

Table 1 describes the study population according to all variables considered in this analysis. Cases and controls had similar distributions according to age group and ethnicity, and small differences for other characteristics, except that a greater proportion of controls had one or more children and a longer duration of oral contraceptive use. Among EOC subgroups, invasive cases were more likely to be postmenopausal and have a family history of ovarian or breast cancer compared with controls, while borderline cases were younger and more likely to be premenopausal, less educated, and have more pack-years of smoking compared with controls.

Just over half of both cases and controls participated in any shift work, and similar distributions were observed for cases and controls for participation in individual shift work patterns (Fig. 1). Table 2 shows the main occupations in which shift work was recorded. Medical, dental, veterinary, and related workers (13.0% of all shift workers); cooks, waiters, bartenders, and related workers (10.2%); and bookkeepers, cashiers, and related workers (9.4%) were the top three shift work occupations. When examined according to shift times and schedules, professional nurses were common across any shift times/schedules, and particularly for fixed night shifts. Among fixed evening shifts, "authors, journalists, and related writers" was the most common occupation group and among rotating shifts, "salespeople, shop assistants, and sales demonstrators" was the most common occupation group.

Table 3 displays associations for cumulative years of shift work with overall EOC risk as well as risk by tumor behavior. For EOC overall, the OR (95% CI) for the highest category of cumulative years of exposure to any shift work (i.e., > 12 years) versus never exposed to shift work was 1.21 (0.89–1.63); however, a monotonic dose–response relationship pattern was not observed. Similarly, no strong pattern of association was observed for shift work variables defined according to shift times and schedules (Table 3). These adjusted ORs did not appreciably differ when occupational history for 2, 5, and 10 years prior to referent date was excluded (results not shown). When never shift workers were removed and the lowest shift work category was used as a reference, the ORs reflected the same pattern of associations seen in Table 2 (results not shown). When cumulative years of exposure to any shift work was categorized as ever versus never shift work, the adjusted OR (95% CI) for overall EOC risk was 1.00 (0.99–1.01). When categorized using cutoffs from breast cancer studies, the adjusted ORs (95% CI), compared with never shift work, were 0.96 (0.75–1.23) for <15 years, 1.26 (0.87–1.82) for 15–29 years, and 1.20 (0.72–2.01) for >29 years. When we restricted the analysis to women who have held at least one job outside of the home, to address the fact that workers may be generally healthier, the ORs were virtually unchanged (results not shown). Associations for invasive and borderline tumors separately did not appreciably differ from each other, nor from what was seen for all EOCs combined (Table 3). When cases were restricted to HGSC, the adjusted ORs (95 CI %), compared with never shift work, were 1.29 (0.86–1.94) for <5 years versus never shift work, 0.75 (0.48–1.17) for 5–12 years versus never shift work, and 1.40 (0.97–2.04) for >12 years.

When focusing on night shift work, the observed ORs did not significantly differ from the null value for different levels of both average number of night shifts per month and average number of consecutive night shifts per month, both compared with never shift workers (Table 4). When examined according to chronotype, a positive association between shift work and EOC overall was observed among women identified as having "morning" chronotypes, which was statistically significant for the highest category of cumulative years of any shift work, while among women identified as having "evening" chronotypes an inverse association was observed, also statistically significant for the highest category of cumulative years of any shift work (Table 5); however, this difference in ORs for women with "morning" versus "evening" chronotypes was not statistically significant. Inverse associations for the highest cumulative shift work years versus never among women with "evening" chronotypes were also suggested for the shift patterns of evening shift work only (OR = 0.63; 95% CI: 0.40–1.52) and rotating shift work only (OR = 0.67; 95% CI: 0.26–1.77). Associations between cumulative years of any shift work and EOC risk did not significantly vary between premenopausal and postmenopausal women (Table 5).

Discussion

In this population-based case–control study, we did not observe evidence of an association between cumulative years of shift work, defined as any shift work as well as according to shift times (ever night shift work, evening shift work only) and schedules (rotating shift work only, fixed shift work only), and overall EOC risk. Associations for invasive and borderline EOC were similar to that observed for EOC overall. The OR for the highest level of any shift work and HGSC suggested a marginally significant increased risk, but as for the associations of EOC overall and for invasive and borderline cancers separately, the ORs across categories were nonmonotonic. When associations were examined according to chronotype or menopausal status, we did not observe statistically significant differences in associations between cumulative years of shift work and risk of EOC overall. Nonetheless, there was some suggestion that a positive association was specific to women with a "morning" chronotype while among women with an "evening" chronotype, shift work was associated with a reduced EOC risk.

To date, four studies have investigated the specific relationship between shift work and EOC risk (18). A null association was reported in a retrospective cohort study with exposure defined according to census reported job titles linked to a job-exposure matrix defining the percentage of shift workers in each job title (22). Similarly, there was no strong evidence of an association in a prospective cohort study of rotating shift work with night shifts among nurses (21), while in another cohort study a positive association was observed between current rotating work at baseline and fatal ovarian cancer (20). Most similar to ours is the population-based case–control study by Bhatti and colleagues (19) that based exposure on an assessment of lifetime occupational history and enrolled women in similar calendar years. In that study, ever night shift work was associated with increased risks of invasive and borderline EOCs (19). However,

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Table 1. Characteristics of PROVAQ study participants, *n* (%)

	Controls ($N = 906$)	Full case group ($N = 496$)	Invasive cases ($n = 362$)	Borderline cases ($n = 134$)
Age (years)				
<45	116 (12.8)	63 (12.7)	26 (7.2)	37 (27.6)
45 to <55	212 (23.4)	129 (26.0)	97 (26.8)	32 (23.9)
55 to <65	294 (32.5)	162 (32.7)	122 (33.7)	40 (29.9)
65	284 (31.3)	142 (28.7)	117 (32.3)	25 (18.7)
Menopausal status[a,b]				
Premenopausal	291 (32.1)	161 (32.5)	105 (29.7)	56 (41.8)
Postmenopausal	589 (65.0)	323 (65.1)	249 (70.3)	74 (55.2)
Self-reported ethnicity[a]				
French Canadian	607 (67.0)	337 (68.1)	244 (67.6)	93 (69.4)
Other European ancestry	216 (23.9)	115 (23.2)	85 (23.5)	30 (22.4)
Other/mixed ancestry	82 (9.1)	43 (8.7)	32 (8.9)	11 (8.2)
Family history of cancer in first-degree female relatives[a]				
Ovarian	22 (2.4)	26 (5.2)	22 (6.1)	4 (3.0)
Breast	146 (16.1)	89 (17.9)	77 (21.3)	12 (9.0)
Education level				
High school	281 (31.0)	191 (38.5)	134 (37.0)	57 (42.5)
College/technical	277 (30.6)	144 (29.0)	107 (29.6)	37 (27.6)
University, undergraduate	348 (38.4)	161 (32.5)	121 (33.4)	40 (29.9)
BMI (kg/m^2)				
<18.5	36 (4.0)	25 (5.0)	17 (4.7)	8 (6.0)
18.5 to <25	423 (46.7)	218 (44.0)	161 (44.5)	57 (42.5)
25 to <30	277 (30.5)	139 (28.0)	100 (27.6)	39 (29.1)
30	170 (18.8)	114 (23.0)	84 (23.2)	30 (22.4)
Parity (full-term births)				
Nulliparous	197 (21.8)	166 (33.5)	114 (31.5)	52 (38.8)
1	160 (17.7)	102 (20.6)	77 (21.3)	25 (18.7)
2	354 (39.1)	156 (31.4)	115 (31.8)	41 (30.6)
3	194 (21.4)	72 (14.5)	56 (15.5)	16 (11.9)
Breastfeeding duration (months)				
Never	475 (52.4)	323 (65.1)	234 (64.7)	89 (66.4)
0 to <6	184 (20.3)	88 (17.7)	66 (18.2)	22 (16.4)
6	247 (27.3)	85 (17.2)	62 (17.1)	23 (17.2)
Oral contraceptive use (years)[a]				
Never	172 (19.0)	107 (21.7)	90 (24.9)	17 (12.9)
0 to <2	158 (17.4)	94 (19.0)	65 (18.0)	29 (22.0)
2 to <10	334 (36.9)	195 (39.5)	146 (40.3)	49 (37.1)
10	242 (26.7)	98 (19.8)	61 (16.9)	37 (28.0)
History of tubal ligation				
Never	662 (73.1)	387 (78.0)	272 (75.1)	115 (85.8)
Ever	244 (26.9)	109 (22.0)	90 (24.9)	19 (14.2)
Hormone replacement therapy use[a]				
Never	619 (69.2)	325 (66.1)	228 (63.5)	97 (72.9)
Ever	276 (30.8)	167 (33.9)	131 (36.5)	36 (27.1)
Endometriosis[c]				
Never	838 (94.2)	424 (87.2)	310 (87.1)	114 (87.7)
Ever	52 (5.8)	62 (12.8)	46 (12.9)	16 (12.3)
Medically diagnosed infertility				
Never	856 (94.5)	459 (92.5)	333 (92.0)	126 (94.0)
Ever	50 (5.5)	37 (7.5)	29 (8.0)	8 (6.0)
Smoking history (pack-years)[a]				
Never	423 (47.1)	197 (41.0)	155 (43.9)	42 (33.1)
0 to <25	304 (33.8)	189 (39.4)	140 (39.7)	49 (38.6)
25	172 (19.1)	94 (19.6)	58 (16.4)	36 (28.3)
Chronotype[a]				
Morning	379 (41.8)	203 (41.0)	156 (43.2)	47 (35.1)
Intermediate	367 (40.5)	214 (43.2)	152 (42.1)	62 (46.3)
Evening	160 (17.7)	78 (15.8)	53 (14.7)	25 (18.6)

[a]Missing information: family history of cancer (25 controls, 8 cases), self-reported ethnicity (1 control, 1 case), oral contraceptive use (2 cases), smoking history (7 controls, 16 cases), and chronotype (1 case).
[b]Menopausal status was unknown for 26 controls and 12 cases (8 invasive cases, 4 borderline cases).
[c]Endometriosis history unknown for 16 controls and 10 cases (6 invasive cases, 4 borderline cases).

cumulative years of night shift work were not associated with a monotonic dose–response relationship; in particular, relative risks increased with increasing cumulative years except for the highest category where the association was attenuated and null (19). Our results also suggested a nonmonotonic relationship, but the shape was different, with the OR for the highest

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Table 2. Most common shift work occupations in the PROVAQ study population, classified according to the International Standard Classification of Occupations 1968 (ISCO-68), *n* (%)

Occupations[a]	Any shift work (n = 1,663)	Fixed night shift (n = 108)	Fixed evening shift (n = 381)	Rotating shift (n = 1,174)
Medical, dental, veterinary, and related workers[b]	217 (13.0)	34 (31.5)	57 (15.0)	126 (10.7)
Professional nurses[c]	169 (10.2)	30 (27.8)	44 (11.5)	95 (8.1)
Medical doctors[c]	27 (1.6)	—	—	27 (2.3)
Cooks, waiters, bartenders, and related workers[b]	156 (9.4)	22 (20.4)	46 (12.1)	88 (7.5)
Bookkeepers, cashiers, and related workers[b]	151 (9.1)	5 (4.6)	34 (8.9)	112 (9.5)
Salespeople, shop assistants, and related workers[b]	142 (8.5)	<5	12 (3.1)	129 (11.0)
Authors, journalists, and related writers[b]	123 (7.4)	<5	63 (16.5)	59 (5.0)
All other occupations[b]	874 (52.6)	45 (41.7)	169 (44.4)	660 (56.2)

[a]Only occupations with a valid ISCO-68 occupation code were included.
[b]Occupation grouping based on 2 digits of ISCO-68.
[c]Occupation grouping based on 3 digits of ISCO-68.

category in our study suggesting a positive association. The study by Bhatti and colleagues (19) included night shift work only, while ours included all shift work types. Also, our sample size was smaller precluding an analysis of long-term shift work; in fact, our highest category of cumulative years was included within their second highest category where they observed an increased risk (19).

The study by Bhatti and colleagues (19) was also the only other study that examined modification of associations by chronotype and reported a positive association between shift work and ovarian cancer among women self-identified as having a "morning" chronotype but not an "evening" chronotype (19). We also observed a positive association among women with a "morning" chronotype, but we further observed an inverse association between shift work and EOC among women with an "evening" chronotype. Given the relatively small numbers of women in each chronotype strata, this may be a chance finding. However, night shift workers were included in our study population and this observation is coherent with the hypothesis that people with circadian rhythms synchronized to be more active in the evening, such that melatonin peaks later, may adapt better to shift work hours. Chronotype has been observed to modify

Table 3. Multivariable ORs (95% CIs) for the relationship between cumulative years of exposure to any shift work and four shift work patterns and overall, invasive, and borderline EOC

Cumulative years of shift work	Controls (N = 906) n (%)[a]	All cases (N = 496) n (%)[a]	All cases OR[b] (95% CI)	Invasive cases (n = 362) n (%)[a]	Invasive cases OR[b] (95% CI)	Borderline cases (n = 134) n (%)[a]	Borderline cases OR[b] (95% CI)	P_{het}[c]
Any shift work								
Never	437 (48.3)	231 (46.6)	1.00 (ref)	171 (47.2)	1.00 (ref)	60 (44.8)	1.00 (ref)	0.65
<5	146 (16.1)	93 (18.8)	1.21 (0.88–1.67)	66 (18.2)	1.22 (0.86–1.73)	27 (20.1)	1.19 (0.71–1.98)	
5–12	168 (18.5)	67 (13.5)	0.74 (0.53–1.03)	44 (12.2)	0.67 (0.46–0.99)	23 (17.2)	0.92 (0.54–1.56)	
>12	155 (17.1)	105 (21.2)	1.21 (0.89–1.63)	81 (22.4)	1.25 (0.90–1.74)	24 (17.9)	1.10 (0.65–1.86)	
P_{trend}[d]			0.75		0.72		0.88	
Ever night shift work								
Never	437 (74.6)	231 (74.8)	1.00 (ref)	171 (75.3)	1.00 (ref)	60 (73.2)	1.00 (ref)	0.48
<5.5	73 (12.4)	40 (12.9)	1.07 (0.70–1.64)	31 (13.7)	1.14 (0.71–1.83)	9 (11.0)	0.85 (0.39–1.84)	
5.5	76 (13.0)	38 (12.3)	0.88 (0.58–1.36)	25 (11.0)	0.80 (0.50–1.32)	13 (15.9)	1.12 (0.58–2.18)	
P_{trend}[d]			0.69		0.56		0.85	
Evening shift work only								
Never	437 (57.7)	231 (55.3)	1.00 (ref)	171 (55.9)	1.00 (ref)	60 (53.6)	1.00 (ref)	0.96
<3	122 (16.1)	82 (19.6)	1.27 (0.92–1.77)	56 (18.3)	1.25 (0.86–1.80)	26 (23.2)	1.33 (0.79–2.25)	
3	198 (26.2)	105 (25.1)	0.98 (0.73–1.31)	79 (25.8)	0.98 (0.72–1.36)	26 (23.2)	0.96 (0.58–1.58)	
P_{trend}[d]			0.92		0.92		0.97	
Rotating shift work only								
Never	437 (62.2)	231 (59.8)	1.00 (ref)	171 (59.8)	1.00 (ref)	60 (60.0)	1.00 (ref)	0.38
<3.5	132 (18.8)	77 (19.9)	1.12 (0.81–1.56)	60 (21.0)	1.23 (0.85–1.76)	17 (17.0)	0.86 (0.48–1.56)	
3.5	133 (19.0)	78 (20.2)	1.06 (0.76–1.47)	55 (19.2)	1.01 (0.70–1.45)	23 (23.0)	1.19 (0.70–2.02)	
P_{trend}[d]			0.64		0.75		0.64	
Fixed shift work only								
Never	437 (83.1)	231 (82.8)	1.00 (ref)	171 (83.4)	1.00 (ref)	60 (81.1)	1.00 (ref)	0.49
<3	31 (5.9)	25 (9.0)	1.50 (0.86–2.63)	19 (9.3)	1.63 (0.89–2.98)	6 (8.1)	1.19 (0.46–3.04)	
3	58 (11.0)	23 (8.2)	0.73 (0.44–1.23)	15 (7.3)	0.64 (0.35–1.17)	8 (10.8)	1.01 (0.45–2.27)	
P_{trend}[d]			0.53		0.41		0.90	

[a]Percentages are based on total number of participants for each shift work exposure group.
[b]Adjusted for age (continuous), education (<high school, high school, college/technical, university undergraduate, university graduate), and parity (nulliparous, 1, 2, 3 full-term births).
[c]P value for heterogeneity between invasive and borderline EOCs.
[d]P value for trend across cumulative years of exposure categories.

Table 4. Multivariable ORs (95% CIs) for the relationship between the average number of night shifts per month and overall EOC risk, and the average number of consecutive night shifts per month and overall EOC risk, among ever night shift workers

Exposure metrics	Cases ($N = 309$) n (%)	Controls ($N = 586$) n (%)	OR[a] (95% CI)
Average number of night shifts per month[b]			
Never	231 (75.3)	437 (75.3)	1.00 (ref)
<12 nights per month	33 (10.7)	70 (12.1)	0.91 (0.58–1.43)
12 nights per month	43 (14.0)	73 (12.6)	1.06 (0.70–1.61)
Average number of consecutive night shifts per month[c]			
Never	231 (79.3)	437 (79.0)	1.00 (ref)
<4 consecutive nights	26 (8.9)	60 (10.8)	0.92 (0.55–1.53)
4 consecutive nights	34 (11.6)	56 (10.2)	1.24 (0.77–2.00)

[a]Adjusted for age (continuous), education (<high school, high school, college/technical, university undergraduate, university graduate), and parity (nulliparous, 1, 2, 3 full-term births).
[b]Two cases and 6 controls had missing data for the average number of night shifts per month.
[c]Fifteen cases and 25 controls were excluded from this analysis, as the question on number of consecutive night shifts per month was added after their study participation; a further 3 cases and 8 controls had missing data for this variable.

associations for shift work with other hormone-sensitive cancers (i.e., breast and prostate cancers; refs. 35–37). The investigation of menopausal status as a potential effect modifier allowed us to examine whether different hormone profiles may differentially affect exposure to shift work in association with ovarian cancer. Similar to the only other ovarian cancer study that examined associations by menopausal status (21), we observed that ORs did not vary according to menopausal status.

The collection of detailed shift information for each job held by participants enabled the analysis of individual shift work patterns defined according to shift times and schedules. Although we did not observe evidence of associations, these analyses allowed us to address the hypothesis that different shift work patterns may contribute to varying degrees of circadian disruption (23, 24). The frequency and intensity of night shift work have only been analyzed for breast cancer risk, where two studies reported that night shift workers working more frequent and intense schedules have increased risks (35, 38). Our study included a small number of night shift workers with a high frequency or high intensity of night shifts, thus the OR estimates for these analyses were imprecise.

Despite the inclusion of almost 500 cases and the fact that a large proportion of the PROVAQ study population was exposed to shift work, relatively small numbers were exposed to long-term shift work. Thus, if an association exists for long-term shift work (e.g., >25 years) as seen in some breast cancer studies (30), we would not have been able to detect this. We believe that if there were errors in recounting shift work history, this would likely have affected cases and controls equally as participants were not directly asked to report their previous shift work, rather they were asked about several job details, including work patterns involving shifts, after they had first listed all jobs in their history with the aid of a life events calendar. According to one study, in comparison with individual payroll data, the reporting of shift work experiences with night shifts demonstrated high sensitivity (>90%) and specificity (>92%), and questions on shift work experiences without night shifts showed low sensitivity (62%) and moderate specificity (87%; ref. 39). Chronotype may have been misclassified in our study due to self-report, compared with other studies that utilized tools such as the Munich ChronoType Questionnaire (40), which takes into account temporal preferences on work and nonwork days, specific sleep and activity times, and outdoor light exposure in the determination of an individual's chronotype. However, the degree of misclassification may be minimal as one study has demonstrated that self-reported chronotype is highly correlated to the determination of chronotype using a validated questionnaire (41). Furthermore, our results suggest that associations varied by chronotype in a direction consistent with the hypothesis that women with "evening" chronotypes may be better adapted to shift work hours compared with women with "morning" chronotypes.

Table 5. Multivariable ORs (95% CIs) for the relationship between cumulative years of exposure to any shift work and overall risk of ovarian cancer, by chronotype and menopausal status

	Cumulative years of exposure to any shift work				P	
	Never	<5	5–12	>12	Trend[a]	Interaction[b]
By chronotype						
Morning type						0.29
#cases/#controls	101/207	36/55	24/66	42/51		
OR[c] (95% CI)	1.00 (ref)	1.43 (0.87–2.34)	0.82 (0.48–1.39)	1.64 (1.01–2.65)	0.16	
Intermediate type[d]						
#cases/#controls	99/190	44/70	36/75	43/62		
OR[c] (95% CI)	1.00 (ref)	0.82 (0.42–1.61)	1.04 (0.52–2.11)	0.74 (0.38–1.45)	0.47	
Evening type						
#cases/#controls	30/40	13/21	7/27	20/42		
OR[c] (95% CI)	1.00 (ref)	0.56 (0.21–1.51)	0.36 (0.12–1.10)	0.37 (0.15–0.88)	0.02	
By menopausal status						
Premenopausal						0.67
#cases/#controls	67/129	45/62	25/67	24/33		
OR[c] (95% CI)	1.00 (ref)	1.45 (0.89–2.39)	0.67 (0.38–1.16)	1.32 (0.71–2.45)	0.97	
Postmenopausal						
#cases/#controls	161/294	46/81	38/96	78/118		
OR[c] (95% CI)	1.00 (ref)	0.71 (0.37–1.35)	0.12 (0.55–2.25)	0.87 (0.43–1.76)	0.80	

[a]P_{trend} across cumulative years of exposure categories.
[b]$P_{interaction}$ using the likelihood ratio test to compare regression models with and without interaction terms.
[c]Adjusted for age (continuous), education (<high school, high school, college/technical, university undergraduate, university graduate), and parity (nulliparous, 1, 2, 3 full-term births).
[d]Intermediate chronotype combined women reporting that they were "more morning than evening" or "more evening than morning" people.

Because the participation rate among controls was 56% and information collected from eligible nonparticipating controls indicated they were older and had a lower level of education (31), participating controls may not have accurately represented the study base with respect to shift work prevalence. In particular, education level is associated with shift work participation, as shift work is more common in occupations that provide services 24 hours per day, such as healthcare and social assistance, retail trades, and accommodation and food services (42). The adjustment for education level in our analyses reduced the impact of potential selection bias due to differential participation according to education level (43). Although we considered a variety of confounders in our directed acyclic graphs, uncontrolled confounding from unknown factors related to shift work participation cannot be ruled out.

In summary, this study does not support an overall association between shift work exposure and EOC risk. However, our results suggest that chronotype should be considered in studies of shift work as an exposure. As shift work is a prevalent exposure that is a probable human carcinogen, the examination of the organization of shift work, such as according to shift times and schedules, may lead to an increased understanding of the role on cancer risk.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

Authors' Contributions

Conception and design: J. Siemiatycki, K.J. Aronson, A. Koushik
Development of methodology: J. Siemiatycki, K.J. Aronson, A. Koushik
Acquisition of data (provided animals, acquired and managed patients, provided facilities, etc.): J. Arseneau, L. Gilbert, W.H. Gotlieb, D.M. Provencher, A. Koushik
Analysis and interpretation of data (e.g., statistical analysis, biostatistics, computational analysis): L. Leung, A. Grundy, J. Arseneau, L. Gilbert, W.H. Gotlieb, K.J. Aronson, A. Koushik
Writing, review, and/or revision of the manuscript: L. Leung, A. Grundy, J. Siemiatycki, L. Gilbert, W.H. Gotlieb, D.M. Provencher, K.J. Aronson, A. Koushik
Administrative, technical, or material support (i.e., reporting or organizing data, constructing databases): L. Leung, A. Grundy, A. Koushik
Study supervision: W.H. Gotlieb, A. Koushik
Other (recruitment of patients, interpreting results and critical review of manuscript): L. Gilbert
Other (co-supervision with A. Koushik and L. Leung for Masters of Science in Epidemiology): K.J. Aronson
Other (principal Investigator of the PROVAQ project): A. Koushik

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Exhibit B

UNITED STATES DISTRICT COURT
DISTRICT OF NEW JERSEY

IN RE JOHNSON & JOHNSON TALCUM POWDER PRODUCTS MARKETING, SALES PRACTICES, AND PRODUCTS LIABILITY LITIGATION

THIS DOCUMENT RELATES TO ALL CASES

MDL NO. 16-2738 (FLW) (LHG)

RULE 26 EXPERT REPORT OF JACK SIEMIATYCKI MSc, PhD

J Siemiatycki

Date: November 16, 2018

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Jack Siemiatycki MSc, PhD

EXPERT REPORT OF JACK SIEMIATYCKI MSc, PhD

On

TALCUM POWDER USE AND OVARIAN CANCER

Jack Siemiatycki, MSc, PhD, FCAHS

106 Columbia Avenue

Westmount, Quebec, Canada

November 16, 2018

TABLE OF CONTENTS

1. My mandate

I have been retained to assess the epidemiologic evidence regarding the **general causation** between perineal (or genital) use of talcum powder products and risk of ovarian cancer. The question is: "Can application of talcum powder products in the perineal region cause ovarian cancer?"

All of my opinions in this report are stated to a reasonable degree of scientific certainty.

2. My credentials, expertise and experience

I am a tenured Professor of epidemiology at the University of Montreal and an Adjunct Professor of epidemiology at McGill University in Montreal. I have received prestigious national research awards in Canada, such as National Health Scientist Salary Award, Medical Research Council Distinguished Scientist Award, Canada Research Chair in Environment and Cancer and, currently, I hold the Guzzo-Cancer Research Society Chair in Environment and Cancer. I am an elected fellow of the Canadian Academy of Health Sciences. I was awarded a lifetime achievement award by the Canadian Society for Epidemiology and Biostatistics, the premier professional organisation in our discipline.

Trained in statistics and in epidemiology, I have devoted most of my research career to investigating links between environmental, occupational and lifestyle factors and various types of cancer. My research has been both substantive – namely, looking at particular factors and their possible relationship to particular cancers - and methodological – namely, exploring how to evaluate and enhance the validity of epidemiologic research through various prisms: study design, data collection methods and statistical analysis. Of my approximately 250 research publications, about one quarter would be considered to have methodological focus.

I have held various leadership positions, including the elected presidency of the Canadian Society for Epidemiology and Biostatistics, and elected membership on the Board of the American College of Epidemiology. I have been invited to serve on over 160 Boards, Scientific Councils and Expert Panels for a host of governments, universities or research agencies. Examples include: Board of Directors of the Canadian National Cancer Institute, member of expert panel tasked with recommending priorities for action under the

Canadian Environmental Protection Act, member of external peer review panel of the Epidemiology branch of the US National Cancer Institute (NCI), member of two different expert advisory bodies to research projects at the NCI, consulted by President Clinton's Cancer panel, member of external peer review panel for the Helmholtz German national medical research agency, Chair of the Scientific Council of the largest prospective study of causes of cancer being conducted in France, and others of that nature.

I have been associate editor of the American Journal of Epidemiology and the International Journal of Environmental Health. In addition, I have served as reviewer for about 20 journals. I have served as a chair and as a member of grant review panels for major Canadian scientific funding agencies.

My research programme has been well funded by Canadian funding agencies for over 35 years. I have conducted research and published on the carcinogenicity of a large number of agents in the occupational environment (e.g. asbestos, silica, welding fumes) and in the general environment (e.g. smoke from wood stoves, urban air pollution) and lifestyle factors (e.g. smoking, alcohol, use of cell phones).

I have taught and supervised epidemiology students and many of my former trainees are now faculty members in universities around the world.

I have had a long association with the International Agency for Research on Cancer (IARC). IARC is the premier institution in the world for cancer epidemiology and for environment and cancer research. It has several mandates, including the organisation and compilation of standardised high quality data on cancer incidence around the world, the conduct of original research, and the evaluation of the carcinogenicity of different agents with which humans come into contact. The latter is achieved through a process that involves identifying chemical or physical agents for evaluation, convening specially selected international expert panels that are mandated to review all pertinent evidence on the topic and write a thorough review culminating in an evaluation of whether the agent(s) are human carcinogens. Since the inception of this program in 1971, there have been about 120 meetings held and approximately 1100 agents have been evaluated.

A particular point of pride for me is that over the years, research results from my team have been cited as part of the information base on 69 of the 1100 agents that have been evaluated, probably making my team the most cited epidemiology team in the history of the IARC Monograph program.

My association with IARC began when I did a post-doctoral fellowship there in 1977-79. Over the intervening years I have collaborated with scientists at IARC on various research projects. I was a member of the 18-member Scientific Council of IARC from 2006 to 2010 including two years as elected Chairman of the Council. The Scientific Council oversees all of the scientific activities at IARC; its members are named by the member states of IARC. I have been invited to sit on IARC Monograph international expert panels for 5 of the 60 panels convened in the past 25 years. One of the IARC Monograph panels of which I was a member was tasked with evaluating: "Carbon black, titanium dioxide and non-asbestiform talc." Out of the 16 invited experts who participated in the meeting as members of the Working Group, I was selected to chair the meeting.

Subsequent to the IARC meeting and the report of the meeting, a small subgroup of members of the IARC Working Group, of which I was a member, conducted and published a meta-analysis of the results of the studies that had been available to the IARC Working Group (Langseth, 2008)

Although I have not personally produced original data collection studies on the topic, I am well qualified to review the epidemiologic evidence. I have participated in two published reviews of the issue. The methodologic expertise and analytical skills required to critically review and evaluate such evidence is generic to the vast area of environmental epidemiology of cancer. I am routinely asked by journals and grant agencies to provide expert opinions on topics for which I have not produced original data collection studies, but that are within the purview of my expertise. The invitation by IARC to chair the meeting at which talc was evaluated is testimony to the fact that my competence and expertise in this matter are internationally recognized by peers. I do not claim expertise in various adjoining domains that inform this issue, including physiology, pathology, clinical oncology, experimental toxicology, geology and mineral chemistry. However, I do have the

expertise and skill to assimilate information that is provided by experts in these areas. I have previously submitted a report on my review of the evidence regarding talcum powder products and ovarian cancer in October 2016.

I have previously served as an expert witness for plaintiffs in one U.S. court case, and that was a talc litigation in Los Angeles in 2017. (Eva Echeverria, BC628228, Johnson and Johnson Talcum Powder Cases, CA JCCP No. 4872), and I testified that the genital use of talcum powder products can cause ovarian cancer.

I have served as an expert witness in two Canadian court cases, neither having to do with talc or hygiene powders or ovarian cancer. One case dealt with a class action lawsuit on behalf of a town in Canada adjoining a Canadian military base where there had allegedly been a spill of trichloroethylene that seeped into the water table of the town. The residents claimed that the contamination had caused cases of cancer. I was an expert for the defence, the Canadian government, and I testified in 2012. (Province of Quebec Superior Court file 200-06-000038-037).

The other case was a class action on behalf of Quebec residents who contracted cancer and had been smokers, claiming that the tobacco companies were responsible for their diseases. I was an expert for the plaintiffs and I testified in 2014. (Province of Quebec Superior Court file 500-06-000076-980).

In my work as an expert for legal cases, my time is billed at the rate of $450 per hour for research, report preparation, communications with counsel, participation in depositions, and testimony in court.

3. Overview of my methodology

The basis of my opinions derive from my education, training, experience, research and what is accepted within the community of leading scientists practicing in the field of epidemiology. My opinions are based on my review of the relevant materials, published in the scientific literature and/or produced in this case; including internal company documents, as well as relevant depositions, reports and testimony in the talcum powder product litigation. To reach my conclusions, I have employed the same scientific

methodology and rigor that I use in my research, in my publications and in the consulting and advising that I carry out on behalf of governments, public health agencies and research institutes. This includes a review of the relevant published literature, expert judgment to assess the quality and meaning of the various studies that were reviewed, and syntheses of the available evidence and any other pertinent medical and scientific evidence of which I am aware. The methods I used to derive and present my opinions are those used in general in the assessment of causal relations in medicine and public health, and more specifically in epidemiology. The methods are based on the experience and insight I have accumulated over 40 years of research, consulting, reviewing and student supervision, from discussions and interactions with leading epidemiologists, service on multiple IARC panels, and from reading evolving ideas in the scientific literature, including such seminal works as Bradford Hill's (Hill 1965) writings on assessing causality.

My opinions may be further supplemented and refined, subject to results that may come from further medical and scientific study and research and the continued review of additional information and discovery materials produced in this litigation.

4. The science of epidemiology

This section is designed to provide a non-specialist reader with information and definitions about epidemiology and biostatistics that are needed to understand the basis of my evaluation on talcum powder products and ovarian cancer. I do not present in this section the actual data and evidence regarding talcum powder products and ovarian cancer.

Epidemiology is the science of occurrence of diseases in human populations. It is concerned with the patterns of disease occurrence and also with identifying the factors that influence disease occurrence. These two sets of concerns are sometimes referred to respectively as descriptive epidemiology and analytic epidemiology. The first addresses such issues as the incidence of the disease in different geographic areas, in different time periods, or at different ages and sexes. The second addresses more focused questions on the specific environmental and/or lifestyle and/or genetic factors that might influence the incidence of disease.

The word "epidemiology" has the same etymologic roots as the word "epidemic", which signifies that, initially, epidemiology grew out of the study of epidemics. Such epidemics were often of a microbial origin (e.g. viruses, bacteria, parasites). But increasingly in the 19th and especially in the 20th century, it became clear that the etiology (i.e. causation) of chronic diseases such as cancer could also be elucidated by studying their patterns of occurrence.

While there were many studies carried out in the early to mid-20th century that we would now qualify as epidemiological in nature, the discipline of epidemiology and its methods started to become formalized in the 1950's and 1960's. There are now departments of epidemiology in most large universities that have health science research and teaching activities and there are many national and international societies of epidemiology.

Epidemiology is characterized by its mainly observational and non-experimental approaches. It is a discipline that is not primarily based in the laboratory; rather it is based in society. That is the source of its strength, and its weakness. Because it deals with people in the reality of their lives, it is the most pertinent approach to understanding the links between people's lifestyles and environments and their health and disease. However, because it is based in society, it by necessity confronts the extreme complexity of human lifestyles, environments and diseases. And because we cannot experiment with people's lives, we cannot control the conditions in which people are exposed. The methods of epidemiologic research are complex and differ from study to study. Statistical methods play an important role in trying to tease out the role of different variables and in determining whether the observed results may be attributable to chance, to bias or to real effects of putative risk factors. It is usually necessary to assemble evidence from several data-collection studies on a given topic before being able to draw inferences about causality.

4.1 Some basic measures and notions used in epidemiology

In this section I will review a number of concepts that need to be understood in order to properly understand my review of the evidence regarding talc powder and ovarian cancer. It is intended for readers who may not be expert in epidemiology. In this section I will not necessarily tie the concepts and definitions to the talc-ovarian cancer issue; that part will

be left for later. For now, I am simply introducing the non-epidemiologist reader to terminology and concepts with which she/he may not be very familiar.

Prevalence of disease. The prevalence of a disease refers to the proportion of a population who are living with the disease at any given point in time.

Incidence of disease. The incidence of a disease refers to the proportion of a population who are newly diagnosed with the disease during a certain period of time. The bridge between incidence rate and prevalence rate is the average duration of the disease, or how long people live with it before they are cured or pass away. In fact, while incidence and prevalence are foundational concepts in epidemiology, it is only incidence that figures prominently in the evaluation of carcinogenicity of talc.

Risk of disease. The risk of disease is a term that can refer to incidence or prevalence. The meaning should be clear from the context in which it is used. For studies of cancer, it almost always refers to incidence of disease. This is the way I will use the term in this report.

"Cause" of disease. A cause of a disease is any agent or characteristic (environmental, lifestyle or genetic) that increases the probability of getting the disease or it may simply advance or hasten the onset of the disease. It may act alone or it may act in concert with other factors over a lifetime to cause the disease. It may act immediately (e.g., cyanide as a cause of poisoning; lack of seat belt use as a cause of car accident mortality) or it may take many years for the effect to become manifest (e.g., lack of physical activity as a cause of obesity). There may be many different causes for the same disease. (See explanation of Multifactorial Etiology below.)

Risk factor. As defined in the Dictionary of Epidemiology (Last 2001), a risk factor is an aspect of personal behavior or life-style, an environmental exposure, or an inborn or inherited characteristic, that, on the basis of epidemiologic evidence, is known to be associated with a health-related condition. The term *risk factor* is used rather loosely and depending on the context it can refer to a factor that directly causes a disease or a factor that is a strong marker for the proximal cause of the disease. As it is often used, I will

mainly use the term "risk factor" as a synonym for the noun "cause" of the disease. (eg. "Smoking is a risk factor for lung cancer.")

Association. As defined in the Dictionary of Epidemiology (Last 2001), association refers to the degree of statistical dependence between two or more events or variables. Events are said to be associated when they occur more frequently together than one would expect by chance. Association does not necessarily imply a causal relationship.

Risk among unexposed (R_u) refers to the risk of disease among persons who are not (or were not) exposed to the agent under investigation. In this case, it would refer to the risk of getting ovarian cancer among women who *have never* used talc in the perineal region.

Risk among exposed (R_e) refers to the risk of disease among persons who are (or were) exposed to the agent under investigation. In this case, it would refer to the risk of getting ovarian cancer among women who *have* used talc in the perineal region.

Relative Risk: $RR = R_e/R_u$ = Risk among exposed/Risk among unexposed

When RR > 1.0, it indicates that exposure to the agent increases the risk of developing the disease. When RR < 1.0, it indicates that exposure to the agent prevents the disease.

When RR = 1.0, it indicates that the exposure to the agent has no bearing on the risk of getting the disease.

95% Confidence interval (95% CI). This refers to the precision of an estimate of a parameter. When we estimate the 95% CI for the RR, we are approximately saying that we are 95% certain that the true parameter underlying the study is within these limits. (The true interpretation is more subtle.)

Statistical significance of an association: Statistical significance is a measure of the departure of a set of data from some null hypothesis. Most commonly in epidemiology, the null hypothesis would state that there is no association between a factor and a disease. The null hypothesis can be operationalized in different ways, such as that the RR = 1.0, or that there is no trend between the degree of exposure and the RR. Once a study is conducted, the results can be compared with the expected results based on the null hypothesis, and the discrepancy from the null hypothesis is measurable with probabilities. This is done

either by computing a p-value or a confidence interval. If the p-value is very small or the confidence interval does not include the null value, then we say that an observed association between the putative risk factor and the disease is unlikely to be due to chance alone.

It is important to note that while statistical significance is a tool for assessing whether an observed association is attributable to chance alone, it is not a very effective tool for establishing the absence of an association. That is, <u>the absence of statistical significance is not tantamount to proof of the absence of an association</u>. The absence of statistical significance can be due to the true absence of an association, but it can also be due to the study not having sufficient statistical power or to bias or confounding in the research methods. Furthermore, it should be noted that the conventional dichotomization of results as "statistically significant" or not, based on a particular cutpoint on the p-value scale (eg. p = 0.05), is a gross simplification. The compatibility of the data with the null hypothesis of no association is in truth on a continuous scale and the dichotomization is arbitrary and potentially misleading, especially when the observed p-value is close to the arbitrary cutpoint.

In practice, epidemiologists have been moving away from using and reporting p-values and statistical significance, as it has become clear that the main contribution of an individual study is to provide an estimate of the relative risk and its range of plausible values, embodied in a confidence interval.

<u>Cohort studies and case-control studies:</u> Epidemiologic research projects can take many different forms. The two most common types of analytic epidemiologic studies are cohort studies and case-control studies. (Rothman, Greenland, & Lash 2008)

In a cohort study, it is typical to enrol a large number of subjects, determine which ones are or have been exposed to the factor of interest (e.g., talc) and follow them for some period of time to evaluate whether those who were exposed subsequently experienced different disease rates from those who were not exposed.

In a case-control study, by contrast, we start with people who have the disease under study (e.g. ovarian cancer) and a set of controls who do not have the disease, and we collect data

to determine whether the cases and the controls had different histories of exposure to the factor under study (e.g. talc).

It may be said that a cohort study proceeds from the cause to the effect, whereas a case-control study starts from the effect and backtracks to the cause. There are many variants on these basic designs. These descriptions of these types of study are somewhat simplified.

It is sometimes claimed that a prospective cohort design produces more valid and reliable RR estimates than a case-control study. But this is incorrect as a generalization. The validity and reliability are not determined by the overall architecture of the study, but rather by the specifics of the study, including how the study subjects were assembled, the nature of the variables under study (exposure, disease, confounders), exactly how the information was collected, the statistical power, and so on. There may be many reasons why a particular case-control study is more valid than a particular cohort study.

Relative Risk (RR) and Odds Ratio (OR). The cohort study design leads naturally to the estimation of risk of disease among exposed, and risk of disease among unexposed, and then to the ratio of those two, which is the RR. In case-control studies, because of the way the study samples are selected, it is impossible to estimate the risk of disease or the ratio of the two risks, R_e/R_u. However, under certain conditions which are well met in studies of cancer, it is possible to estimate an approximation of the RR. This is called the odds ratio, referred to as OR. In the rest of this report, I will consider evidence obtained from both cohort studies and case-control studies, and I will refer to the findings of these studies as RRs, even if technically speaking, the results from case-control studies are ORs.

Bias, confounding, effect modification. The aim in an epidemiologic investigation of a putative risk factor is to derive an accurate estimate of the RR between exposure to the agent and the disease at issue. Because the investigator does not control the conditions in which people live and are exposed to different agents and their willingness to participate in research, there are many potential sources of distortion in epidemiologic research. While there are many sources of distortion, they can be bundled into a few large families of sources of distortion.

Bias refers to a systematic distortion in study findings, resulting from the way the study was designed or the way the data are collected. Specific examples of types of bias will be discussed below as they pertain to talc and ovarian cancer.

Confounding is sometimes considered to be a type of bias, and sometimes it is considered a type of distortion on its own. This is merely a semantic distinction. Confounding refers to the situation where the association under study between factor F and disease D is distorted because there is a third factor X which happens to be correlated with F and which is a cause of disease D. For instance if we want to study the association between occupational exposure to talc in mines (factor F) and lung cancer (disease D), we need to be mindful of whether cigarette smoking (factor X) is more common in talc miners than in the rest of the population. Confounding differs from other types of bias in that it depends on relationships among different variables in the population, rather than characteristics of the study design and data collection.

Effect modification refers to the phenomenon whereby a given factor has a different effect in one sub-population than in another. If we study the association between that factor and the disease in the entire population without distinguishing the two sub-populations, we might end up with an estimate of the association that does not convey accurately the association in either sub-population. For instance, if it were the case that a certain genetic characteristic G increases the risk of pre-menopausal ovarian cancer but has no impact on post-menopausal ovarian cancer, then a study of the association between G and ovarian cancer that does not discriminate by menopausal status, would find an RR result somewhere between the null value among post-menopausal women and the true RR value among pre-menopausal women. Depending on the proportions of pre- and post-menopausal women in the sample, the overall RR might be so close to the null, that we might erroneously conclude that there is no association at all. In this example, it might actually be quite simple to detect the effect modification, since age is always recorded and menopausal status is usually recorded and investigators are sensitized to the possible effect modification of female cancers by hormonal status. Other potential effect modifiers may not be so easily available and they might not be on the radar screens of investigators. Effect modification can in some unusual circumstances completely wipe out a true causal

association (as when the agent causes cancer in some people but prevents cancer in others!). But generally, if there is a causal effect of the agent in one stratum of the population and no association in another stratum, and if we fail to stratify the population according to the effect modifier, it will have the effect of producing an overall RR that is lower than it truly is in the sensitive stratum and higher than it truly is in the insensitive stratum.

Effect modification is closely related to and sometimes synonymous with interaction or synergism.

Publication bias refers to the tendency for some evidence never to "see the light of day". Namely, when results are "negative" or "null", it may be that investigators never bother to submit them for publication, or alternatively that editors refuse to publish them. This happens, most likely, when the hypothesis under study is not particularly topical or controversial, and when the study is small.

In this section I have briefly outlined some potential sources of distortion of a typical epidemiologic study. I have done this in a high-level generic way. Below, after presenting results of my review of pertinent literature on powders and ovarian cancer I will return to commenting on the possible impact of such distortions in this body of literature.

Exposure variable and exposure metric

An ***exposure variable*** can be anything that can influence the occurrence or outcome of disease. The term is used for such disparate entities as external components of what we eat, drink, breathe, hear or see and microbiological organisms, chemicals or forms of radiation.

Depending on the nature of the variable, information on an exposure variable can often be ascertained from epidemiologic study participants by questioning them. This is the case for variables like cigarette smoking or use of talc powders. For some variables, like exposure to a virus or to specific air pollutants or occupational chemicals, it is usually necessary to invoke more intensive data collection methods to ascertain exposure.

An ***exposure metric*** signifies a way of defining a variable for statistical analysis. The simplest metric is a binary variable: exposed or unexposed. For most exposure variables, like exposure to talc powder, there can be a very wide range of degree of exposure. And it is pertinent to create more nuanced exposure metrics that take into account the degree of exposure that different people have experienced, metrics such as duration of exposure, intensity or frequency of exposure and even cumulative measures of exposure over long periods of time.

Measurement error. Whenever we are measuring a variable in an epidemiologic study, be it smoking, or weight, or socio-economic status, or blood pressure, or any other variable, it is virtually inevitable that there will be some degree of error in the measurement. There are ways of collecting data that make them more or less likely to involve error, but it is almost impossible to ensure that variables are measured with perfect validity and precision. Even such a variable as the diagnosis of ovarian cancer is subject to differences of opinion among pathologists and oncologists and the presence or absence and the histologic type of tumour is not a guaranteed 100% perfect diagnosis. The ascertainment of the lifetime history of talc exposure by means of an interview with a woman in middle age or later in life is certainly susceptible to the caprices of memory and the way the questions are formulated may influence the validity of respondents' reports of lifetime exposure patterns. It is likely that habits that were performed regularly are more reliably recalled than activities that were sporadic or that only occurred many decades earlier. Similar issues arise for all other variables collected in such studies. We refer to measurement error as random (or non-differential) if the degree of measurement error does not differ between cases and controls in case-control studies or between exposed and unexposed in cohort studies. As a general rule of thumb, it can be asserted that random (or non-differential) measurement error has a predictable distorting effect on the RR. Namely, while there are some rather obscure exceptions, non-differential measurement error tends to attenuate the RR towards the null value of 1.0, and the more measurement error, the greater the attenuation. A full explanation for why this is so is quite technical and can be found in advanced epidemiology textbooks, such as Rothman, Greenland and Lash 2008. A very simple explanation is that the presence of measurement error in assigning exposed vs

unexposed status leads to dilution of both the exposed group and the unexposed group. That is, the ostensible exposed group (i.e. the folks who will be labelled as exposed based on the study data collection) will contain some folks who are truly unexposed and the ostensible unexposed group will contain some folks who are truly exposed. If there really is a difference in risk between the true exposed group and the true unexposed group, this difference will be watered down by the inadvertent inclusion in each group of folks who are really in the opposite group. An analogy is the cross-contamination of two cans of paint. Suppose we have a can of pure white paint and a can of pure red paint. Suppose we have a way of quantifying the difference in color tone between the two paints. Then suppose we take some spoonfuls from the red can and pour them into the white can, and likewise take a few spoonfuls of the white paint and pour them into the red can. Now the color contrast between the two cans has been attenuated. The color contrast in this example is like the relative risk in an epidemiological study which has been attenuated because the exposed and unexposed groups have been cross-contaminated.

Dose-response. It is important not only to assess whether there is an association between a variable and a disease when the variable is defined in a binary (exposed vs unexposed) way, but also when the variable is defined in a quantitative or semi-quantitative way. When we analyse the risk as a function of the degree or duration or intensity of exposure, we refer to this as a dose-response (or exposure-response) analysis. The example of the smoking and lung cancer is instructive about the value of different metrics, though it cannot be assumed that all risk factors act the same way. Studies using the binary metric for smoking (smoker/non-smoker) have been very consistent and persuasive in demonstrating an association between smoking and lung cancer. Further, when data are collected and analysed regarding the degree of smoking, it becomes clear that there is a monotonic dose-response relationship. That is, the more smoking, the higher the risk. And the quantitative metric that manifests the strongest association with lung cancer is the cumulative amount smoked over the lifetime. This is perfectly logical. Since the cumulative exposure metric embodies information on duration and on intensity, it can hardly be less predictive of risk than either of the dimensions alone.

We cannot assume that there is a universal form of a dose-response relationship for every true causal relationship. Most commonly, in toxicology and epidemiology, the relationship between exposure and risk is monotonic; that is, as one increases, so does the other. This can include linear relationships (i.e. where a straight line on a graph describes the relationship) or exponential or many other curvilinear forms. It is also possible that there may be a threshold effect (the risk only becomes apparent after a certain level of effective exposure) or some other non-standard relationship.

Both the qualitative metrics (ever/never) and quantitative metrics (a lot of use compared with a little use) are valid and useful metrics.

Sample size refers to the number of participants in the study. As a generalization, large studies produce more statistically stable and precise estimates than small studies. In fact the stability of estimates or precision of estimates is not a simple function of the number of participants, or subjects, in a study. The precision of estimates depends, among other things, on the type of epidemiologic design.

In a case-control study the main determinants are the numbers of cases and controls and the prevalence of exposure in the two groups; in a cohort study the main determinants are the numbers of participants, prevalence of exposure, and the incidence of the disease of interest over the period of follow-up in the exposed and unexposed groups.

There is sometimes confusion about the notion of sample size when we compare cohort studies with case-control studies. The operational aspect of an epidemiologic study of cancer that most influences the precision of an estimate of RR is not the total number of participants; rather, it is the smaller number between the number of exposed cases of disease and the number of unexposed cases. In a typical prospective cohort study, one might need to enroll 100,000 participants in order to end up with a certain number of cases (say, 500 cases) of the disease of interest (e.g. ovarian cancer). In a case-control design we might only need to enroll around 500 cases and 1500 controls to achieve the same statistical power as would be achieved by a cohort study of 100,000. The formal justification for this assertion is quite mathematical, and has to do with the fact that a sample of a population can give very accurate estimates of the characteristics of an entire

population. Thus, the simple comparison of 100,000 participants in a cohort study and 2,000 participants in a case-control study is in no way a valid marker for the relative statistical power of the two hypothetical studies. There are admittedly other advantages and disadvantages of the cohort vs the case-control design, and reviewers should consider the various aspects before deciding on the relative weight to give to the results of the different studies. But it is definitely not appropriate to merely compare the numbers of participants as an indicator of study validity.

While precision is based on multiple factors and different ones in case-control and cohort studies, there is a parameter which embodies the different factors quite well, and which is common to both case-control and cohort studies, namely, the number of exposed cases. For this reason, in laying out the various study results below, in addition to the relative risk estimates and their confidence intervals, I will show the numbers of exposed cases.

While it may affect the precision of estimates of RR, the size of the study does not in itself systematically affect the estimates of RR. That is, it is not the case that small studies produce systematically exaggerated RR estimates or systematically low RR estimates. However small studies can produce more wildly divergent RR estimates than large ones, in either direction, towards the null or away from the null.

Meta-analysis and pooled analysis: There are two distinct ways that evidence from multiple studies can be combined to derive a new overall statistical summary or synthesis of those studies, a meta-analysis and a pooled analysis. A meta-analysis uses the published results from each study and averages those results using some optimal weighting procedures. In order to implement a meta-analysis it is necessary to find all relevant studies on a topic that have published results in a fairly standardized way. The statistical algorithms typically used to average the results from different studies also provide statistics that evaluate how heterogeneous are the results from the different studies. The interpretation of such heterogeneity statistics is not straightforward. If the results from different studies are homogeneous, it adds to the confidence in the meta-estimate. If they are heterogeneous, it may indicate that the association is really different in different populations, or that there are some methodological characteristics of the different studies

that have influenced the results in different ways. Unless a significant methodological flaw can be identified that has caused the heterogeneity, the best overall estimate remains the meta-estimate.

A pooled analysis is one in which the investigator gets access not only to the published results from different studies, but rather to the individual data of every person in the studies. The latter is harder to achieve because it requires high buy-in and input from the investigators of the original studies; a meta-analysis is much easier to organise. Because a pooled analysis allows for standardization in the definition of variables and statistical models, it can be a more powerful means of summarizing data than the original studies themselves.

Multifactorial etiology of disease. Chronic diseases such as cancer are multifactorial in two distinct ways. On the one hand, each case of disease results from the unfortunate conjuncture of a combination of factors (these might include for example, genetic predisposition, diet, environmental pollutant, occupational exposure, medical intervention, viral infection, lifestyle habits, etc.) which combine over a lifetime to initiate and promote development of the disease. In this sense, each of the factors that are part of the combination for that person was a necessary contributor to the disease process, although it was not sufficient on its own to provoke the disease. Despite the fact that none of the factors were sufficient to produce the disease on their own, each of the contributory factors may be considered to be a cause of the disease. The disease would not have arisen if any of the contributory factors had been absent. This is one meaning of the multifactorial etiology of disease.

The second meaning is that the combination of factors that induce cancer in one person may not be the same as the combination that induces cancer in another person. Indeed, at the population level, there may be many combinations of causal factors for the same disease. Some factors may be common to different combinations. For example, it may be that in one case of lung cancer, the combination of factors included genetics, exposure to air pollution, exposure to radon in the home, and smoking; while in another person, the

combination of factors included genetics, insufficient dietary consumption of anti-oxidants like carotene, exposure to asbestos, and smoking.

Some characteristics of carcinogens and epidemiologic research on cancer: The following characteristics of most known carcinogens provide a framework for some of the thinking behind the design and interpretation of epidemiologic studies of cancer.

- There is typically a long induction period between exposure to a carcinogen and appearance of the disease. Thus, if a study has not allowed for a sufficient passage of time between the exposure and the disease, the result may report that there is no risk, where in fact there is a risk, but insufficient time has elapsed to make the risk visible.
- There is variability in the carcinogenic potency of different carcinogenic agents; some induce much greater relative risks than others.
- For any given carcinogen, the degree of risk due to exposure generally increases as the exposure level increases, but the shape of the dose-response curve may differ from one carcinogen to another.
- Most known human carcinogens were first discovered as such either by means of astute observation of a clinician noticing a cluster of cases among people who shared a common characteristic (such as working in a particular workplace) or by means of epidemiological research. In most cases, there was no known mechanism to explain the association at the time. Where the mechanisms have been elucidated, they were usually discovered subsequent to the epidemiologic demonstration of a causal relationship. (Siemiatycki 2014)

4.2 Bradford Hill "guidelines"

Because of the complexities of epidemiologic research, there has been some concern with how epidemiologic evidence should be used to draw causal inferences. Various authors have written about the types of information that might be considered in assessing whether a body of evidence demonstrates a causal relationship. A set of guidelines, developed in the context of the Surgeon-General's Report on Smoking and Health (1964) and authored by

Bradford Hill in 1965, has achieved a wide consensus in the epidemiologic community as a pedagogical guide. Hill himself referred to these guidelines as "aspects" or "features" or "characteristics" of an association, and warned against treating them as "hard-and-fast rules of evidence that must be obeyed". (Hill, 1965) He deliberately avoided referring to them as "criteria."

Since Hill wrote those thoughts at the beginning of the era of modern epidemiology, without the benefit of decades of practical experience in the way those thoughts were taken up, and how they applied to issues other than smoking and cancer, it is understandable that the practice of evaluation of causality has evolved. A first observation, often overlooked, is that Hill took as a starting point for his writings that chance had been considered as an explanation for the smoking-cancer association and determined to be unlikely. In the historic context of 1964-1965 and the debates around smoking and cancer, this was a reasonable assumption to make, but for any other putative associations, this must be considered. Over the years, respected authors have paraphrased and updated these aspects in various ways, and this will undoubtedly continue. For instance, leading textbooks of epidemiology as well as the Reference Guide on Epidemiology of the Manual on Scientific Evidence (2011) all have different formulations of Hill's guidelines.

In the light of 50 years of practical experience after these guidelines were written, and based on my practical experience of evaluating causality in many forums and on many topics, I would paraphrase (and modernize) Hill's guidelines as follows:

Strength of the association: This can be measured by different parameters, but for cancer studies it is usually measured by the magnitude of the relative risk or odds ratio.

Statistical significance of the association: While this guideline was not explicitly listed by Hill, it is nonetheless in practice an implicit and distinct consideration in assessing causality. If the estimated RR is quite high, indicating a strong association, but is based on a very small study with low precision, this might be solely due to statistical variability. (For instance, when we flip a balanced coin 10 times, we do not always end up with 5 heads and 5 tails. Sometimes, by chance, we may end up with 6 heads and 4 tails. Does this prove that the coin was not balanced?) Evaluating the role of statistical chance as a possible

explanation of the observed association is important. As explained above, the absence of statistical significance is not strong evidence of an absence of a real relationship.

Dose-response relation: If the relative risk increases when the exposure increases, it enhances the likelihood that the observed association is really causal. There are some counter-examples however where the effect is only observed after a threshold of exposure has been crossed. There are various ways to assess whether there is a dose-response relation. Hill pointed out that the main challenge is to establish reliable and measurable quantification of exposure. In studies of lifestyle habits like use of talcum powder products, the most common way is to estimate the RR in increasing categories of exposure metrics such as duration (years) of usage, or intensity of usage (frequency per day or per week or per month), or cumulative amount of usage (a combination of duration and frequency).

Absence of bias: There are many forms of bias that can infiltrate an epidemiologic study. It enhances the likelihood of a true causal association if we can confidently exclude all the plausible sources of bias explanations for the observed findings. This guideline can also be considered as a component of a guideline to consider other possible explanations for the association.

Temporality: It is clear that the exposure should precede the outcome (i.e. the disease). To ascertain whether the cancer was a result of the exposure or the exposure occurred after the cancer onset seems like a simple thing, but sometimes it can be difficult to ascertain with certainty.

Cessation of exposure: It would add to the credibility of the association if it had been demonstrated that subjects who cease exposure to the agent experience reduced risks of disease compared with those who continue to be exposed. In practice this is an extremely difficult characteristic to demonstrate, partly because of the difficulty or even ethical impossibility of changing people's habits for scientific experimentation purposes. But occasionally there may be a "natural experiment" wherein large numbers of people cease their exposure and the effects can subsequently be measured in an epidemiologic fashion.

Specificity of the association: It was believed that individual risk factors have specific pathological effects, and Hill posited that if we observe that a given agent is associated with

many different pathologies, it increases the likelihood that these are somehow spurious observations, resting on some type of bias in the studies of that agent. In reality, this Hill characteristic has fallen out of usage in the intervening years with the demonstration that some agents can indeed provoke multiple different pathologies. Examples include cigarette smoking, ionizing radiation and asbestos fibers.

Consistency of findings between studies (or replication of findings): Because epidemiologic research is susceptible to errors from random variability and from different kinds of study biases, before accepting the apparent association as a generalized phenomenon, it is important to see that similar results are replicated in different studies. When these different studies also encompass different study populations in different communities, it enhances the generalizability of the inferences. Generally speaking, the observation of consistent results in different studies adds to the credibility of an inference that there really is a causal relationship.

Coherence with other types of evidence: In the case of tobacco and cancer, it was seen that the historic trend in lung cancer mortality rates in the US and UK followed quite closely the national trends in consumption of tobacco, with a 20 year lag. This was interpreted by Hill as corroboration of the results observed in case-control and cohort studies. Epidemiologic evidence of coherence could conceivably take many forms, and the opportunity to assess coherence is something that is specific to the factor under investigation. Assessment of coherence with historic mortality trends would only be possible in the case of a factor whose exposure in the population changed quite dramatically over time in a way that can be documented, and for which the attributable fraction of the disease due to that factor is very high. This was the “perfect storm” of circumstances that allowed for an assessment of the tobacco-lung cancer association by means of time trend correlations.

Analogy: Hill reasoned that if a factor is somehow similar to another factor that has already been shown to be a risk factor for the disease, then it increases the plausibility of a similar impact due to that putative factor. This is such a vague guideline, with no clear implementation suggestions, that it is not often referred to and rarely implemented.

Biologic plausibility: This guideline can encompass many dimensions of information, including physiology (can the agent or its metabolites reach the organ?), animal carcinogenesis (does the agent produce tumours in experimental animals?), cell studies that reveal mechanistic data, and other biologic information on the toxicology of the agent.

Implementing Hill's guidelines: As Hill himself insisted, sophisticated users of these guidelines do not use them as a formal checklist. He summarized his views as follows:

> « What I do not believe … is that we can usefully lay down some hard-and-fast rules of evidence that must be obeyed before we accept cause and effect. None of my nine viewpoints can bring indisputable evidence for or against the cause-and-effect hypothesis and none can be required as a *sine qua non*. What they can do, with greater or less strength, is to help us to make up our minds on the fundamental question - is there any other way of explaining the set of facts before us, is there any other answer equally, or more, likely than cause and effect? »

The authors of the Reference Guide on Epidemiology of the Manual on Scientific Evidence (2011) clearly stated that Hill's guidelines are not formal criteria, but rather are more in the nature of a memory aid to help us review the evidence about any given causal association. They stated it this way: "There is no formula or algorithm that can be used to assess whether a causal inference is appropriate based on these guidelines."

I have served on many panels to review evidence of causality on one topic or another, including on several IARC Monograph panels that reviewed evidence of carcinogenicity. The IARC process, like the others I have participated in, does not use the Hill guidelines in any rigid formal way. The ideas embodied in Hill's guidelines permeate our thinking about how to evaluate causality, but the operationalization of these guidelines is specific to the problem and to the expert making these determinations. Thus any suggestion that Hill's "aspects" or "features" or "characteristics" of an association should be used as a formal checklist of criteria is simplistic and wrong. To do so would contradict the opinions of experienced epidemiologists, the Manual on Scientific Evidence, and Bradford Hill himself.

In this section, I have laid out and explained the Bradford Hill guidelines in a generic way. Below, in section 8, I will consider how these apply in the context of the talcum powder – ovarian cancer issue.

5. Epidemiologic evidence regarding talc and ovarian cancer

Following some reports in the early 1980's that raised questions about a possible link between use of cosmetic talc powder by women and the risk of ovarian cancer, there were several epidemiologic studies on the topic. By the early 2000's the issue was garnering some attention in the scientific community. The International Agency for Research on Cancer, the premier agency for evaluation of carcinogens, decided to conduct a review of the issue in 2006. Following that review, there have been further studies conducted on the topic.

In the context of a legal action, my mandate is to review all relevant scientific evidence available to date, in order to provide the court with my opinion regarding the link between talc powder exposure and ovarian cancer. The methodology I employed is the same one I have used in my career as an internationally recognized researcher.

5.1 IARC review and evaluation of talcum powder products

As mentioned above in Section 2, the International Agency for Research on Cancer (IARC) is the premier institution in the world for cancer epidemiology and for environment and cancer research. One of its mandates is the evaluation of the carcinogenicity of different agents with which humans come into contact, and this mandate is carried out by the Monograph Programme of IARC. This is achieved through a process that involves identifying chemical or physical agents for evaluation, convening specially selected international expert panels that are mandated to review all pertinent evidence on the topic and write a thorough review culminating in an evaluation of whether the agent(s) are human carcinogens.

In February 2006, there was such an IARC Monograph meeting to evaluate some agents, including talc. The IARC Working Group comprised 16 highly respected and recognized scientists from around the world; I was asked to Chair the Working Group. We reviewed all

the evidence that was available up to that point in time. This certainly included epidemiologic evidence, but it also included evidence from experimental toxicology, physiology, molecular biology and other domains. The IARC Monograph programme has a formal system for classifying agents. The Working Group must classify an agent into one of the following categories:

1 Carcinogen

2A Probable carcinogen

2B Possible carcinogen

3 Not classifiable

4 Not carcinogen

After reviewing the evidence, the panel concluded that talc was a "possible carcinogen", based primarily on evidence regarding the association between dusting powders and ovarian cancer. Here is the definition of this category from the IARC Monograph:

> "Group 2B: The agent is possibly carcinogenic to humans.
>
> This category is used for agents for which there is *limited evidence of carcinogenicity* in humans and less than *sufficient evidence of carcinogenicity* in experimental animals."

This 2B categorization was based on the panel's decision that there was "limited evidence of carcinogenicity in humans", which is in turn defined by IARC as follows:

> "Limited evidence of carcinogenicity in humans: A positive association has been observed between exposure to the agent and cancer for which a causal interpretation is considered by the Working Group to be credible, but chance, bias or confounding could not be ruled out with reasonable confidence."

Subsequent to the completion of the IARC Monograph on talc, a subgroup of the epidemiologists who were on the IARC Working Group, including myself, reviewed the evidence again, but with a view to producing a meta-analysis of the results from the most informative studies conducted to that time. This resulted in the paper by Langseth et al. (2008). This paper was not an IARC publication.

5.2 Information consulted for the present review

In preparation for formulating my current opinions on this topic I assessed, researched, reviewed and consulted a large number of documents, including, but not limited to: all original epidemiological studies published on this topic, all meta-analyses and opinion pieces, experimental toxicology, molecular biology, mechanistic studies, and the IARC Monograph on talc which reviewed all informative studies that had been published before 2006. I was given access to and also reviewed the various expert reports and depositions that have been submitted in various talc cases, either on behalf of the Plaintiff or Defendant, and various internal company documents obtained in discovery.

I systematically reviewed the lists of references of all relevant studies referenced in the IARC report as well as in various meta-analyses and in all recent articles on the topic to identify yet more relevant publications on talc and cancer.

Because some studies have been published in multiple papers and because some papers have included reports on multiple studies, there is not a one-to-one relationship between studies and published papers.

Additionally, I considered evidence regarding the toxicology of talc by reviewing the toxicology evaluation conducted by the IARC Working Group, the summary of talc's putative toxicology referenced in various scientific publications, and the expert reports of various scientific/medical experts in this case.

The central focus of my review is on the epidemiologic evidence.

A complete listing of the documents I consulted, as well as references cited explicitly in this report, is provided in the Bibliography. The Bibliography is in two Parts; Part A comprises all the publications and reports that can be found in publicly available scientific literature. Part B comprises company documents or documents from reports or testimonies of experts.

5.3 My methodology for this review

Table 1 lists the steps I undertook to accomplish my mandate.

5.3.1 Selecting studies for review

To aid in the present assessment of whether or not there is a causal relationship between talcum powder exposure and ovarian cancer, I carried out an up-to-date review of the scientific literature, primarily the epidemiologic literature, concerning the association between use of talc powder and risk of ovarian cancer. This involved meta-analyses to estimate the effect of having ever used perineal powdering, and an assessment of evidence regarding dose-response.

The first task was to find the relevant publications and to set out the distinct pieces of epidemiologic evidence, namely the results of different studies. Based on a number of reviews on the topic of talc and ovarian cancer, including the IARC report, I systematically went through the reference lists to identify all publications that seemed to contain results on the topic. I further conducted a Pubmed search and this did not produce any new informative publications that had not already been identified. In preparation of the meta-analysis, I eliminated from consideration papers that were outside the bounds of what a meta-analysis should contain (i.e. eliminate review articles, commentaries, meta-analyses, and articles that do not really pertain to the issue of perineal talc and ovarian cancer). From the 40 publications that remained, namely those that contained original results on the association between powdering and ovarian cancer, I extracted all results showing RRs between talc powdering and ovarian cancer, and I had these results put into a Filemaker database. This was a value-free exercise. I made no judgement at that stage about relevance or quality of the study or the published results. It was only an attempt to lay out in one "place" the whole of the evidence and to prepare for subsequent analyses. There were over 730 results in this database. On average each publication contained about 18 different RR results of various aspects of talc powder exposure and various types of ovarian cancer. Some contained fewer and some contained many more. (For instance, one study publication contained 180 results, with varying types of ovarian cancer and varying definitions of exposure to powdering.)

In deciding which results to include in a meta-analysis I had to respect the following principles:

- The results have to pertain to the issue of risk of ovarian cancer in relation to use of talc-based powders.
- Where there are sufficient numbers of results to support meta-analyses, there can be meta-analyses for different types of ovarian cancer, and for different routes of exposure to talc-powders.
- In each meta-analysis, each study should only provide one result, so as to avoid double-counting evidence.
- The decision about inclusion of a study should in no way be influenced by whether or not a particular study demonstrated high risks or low risks.

While these seem like simple principles to respect, there were complicating features of the scientific literature:

- Some studies were reported in multiple publications, sometimes the same study subjects were analysed and reported in different ways and sometimes different subsets of the study population were included in different publications. Sometimes the authors fail to clearly enunciate how the data used in one of their papers overlaps with data used in another of their papers from the same study.
- Different studies used different questions about powder use in their questionnaires, and sometimes the same study reported results by different ways of asking about or defining exposure.
- A given study may have presented one result or many results, each addressing a different definition of the talc exposure variable and different way of grouping the ovarian cancer cases.
- Different studies dealt differently with the histologic sub-types of ovarian cancer, sometimes grouping them all, or sometimes separating them, or sometimes reporting both grouped and separate results for different sub-types.

- Different studies used different metrics for analysing powder exposure and estimating its corresponding RR.
- Different studies dealt differently with the challenge of adjusting powder-related risks for possible confounding by other factors.

Decisions had to be made regarding which types of exposure to consider, which types of ovarian cancer to consider, which metrics of exposure to consider and which studies and publications to consider. It is necessary to be rigorous in making such decisions ahead of time, rather than "cherry-picking" results from different studies that appear to support one theory or another.

Appendix Table A1 provides a list of those 40 publicly available publications that have included some original results that might pertain to the association between powdering and ovarian cancer. **Appendix Table A1** shows which publications were included and which papers were excluded from my meta-analyses. For each of the 14 excluded papers, the table also shows the reason. Some papers were excluded because the results did not pertain to ovarian cancer and powdering in the perineal region. Some papers were excluded because the results presented therein were subsumed by a subsequent publication by the same research team or as part of a pooled analysis of multiple studies. Notwithstanding my intention to identify all unique studies and to extract a best "bottom line" result from each study, the nature of the studies and how they were analysed and reported led to many judgement calls. It must be acknowledged that there can be differences of opinion among equally competent and equally well-motivated scientists in how to choose among the different publications and the different results within publications.

Fortuitously, and unbeknownst to me at the time, two other sets of investigators (Berge et al 2018; Penninkilampi et al 2018) carried out separate meta-analyses on this topic at about the same time as I was carrying out mine, and this gives an opportunity to do some cross-comparison of different reviews and meta-analyses. I will comment on these after presenting the results of my meta-analyses.

5.3.2 What were women exposed to in body powders?

Talc has been the main ingredient of body powders used by women over the past century. "Talc particles are normally plate-like. When viewed under the microscope in bulk samples or on air filters, they may appear to be fibers ... Talc may also form as true mineral fibers that are asbestiform; asbestiform describes the pattern of growth of a mineral that is referred to as a 'habit'. Asbestiform talc fibers are very long and thin."(IARC 2010) The structure of platy talc is characterized by a hexagonal sheet arrangement of silicon-oxygen tetrahedral groups in a common plane which creates a double-sheeted structure. These sheets are easily separated which accounts for the "silky" or "smooth" feel of talcum powder products (IARC, 2010). As a mined mineral, the precise chemical and physical characteristics of talc are in part determined by the particular geological formations from which it is extracted. The local conditions can also produce "impurities" in the extracted talc including asbestos, quartz and various metals. It is claimed that cosmetic talcum powder products normally contain >98% talc (Zazenski *et al.*, 1995) but the purity may have been lower in the past. (IARC 2010) When I refer to talc or talcum powder products in this report, I am referring to commercially available talcum powder products and all constituent elements contained in those products.

Asbestos is a commercial term that comprises six minerals that occur in the asbestiform habit: actinolite, anthophyllite, chrysotile, grunerite, riebeckite and tremolite. Similarly to talc, these six minerals can occur in a non-asbestiform habit. Some types of asbestos are found in the same geological formations as talc.(IARC 2010)

By the 1970's it was reported that asbestos fibers were found in commercial talcum powder (Cralley 1968; Rohl 1976), though there was some doubt expressed regarding the quantification of the exposure and the ability to discriminate between asbestiform and non-asbestiform talc. (Krause 1977; IARC 2010) The talc industry was constrained to remove asbestos from talcum powder products. Representatives of the industry have claimed that talcum powders were free of asbestos fibers since the 1980's (Hopkins 2018; Pier 2018), but this assertion has increasingly come under doubt as number of labs have

reported finding asbestos fibers in talcum powder products. (Blount 1991; Paoletti 1984; Gordon 2014; Longo et al 2017; Longo et al 2018; Blount deposition 2018; Pier deposition 2018) These various studies that have reported finding asbestos in historic talcum powder samples have been challenged by other reports that failed to find meaningful amounts of asbestos in historic talcum powder samples. (CIR 2013; Anderson 2017) These various findings and opinions are somewhat complicated by the fact that both talc and asbestos have varied chemical and physical characteristics and various methods can be used to measure them.

What is clear is that asbestos, and all forms thereof, has been evaluated to be carcinogenic. It has long been recognized that inhalation of asbestos carries with it a risk of lung cancer and of mesothelioma, a cancer of the lining of the lungs, as well as larynx cancer. What has only recently been recognized is that women who are exposed to asbestos experience an excess risk of ovarian cancer. (Straif 2009; IARC 2012) This conclusion was based on five studies; a subsequent meta-analysis reported that the RR of ovarian cancer among asbestos-exposed women was a highly statistically significant 1.77 (1.37-2.28).(Camargo 2011) The route of exposure that generates risk of ovarian cancer among women exposed to asbestos is not clear, but inhalation and migration of asbestos particles to the ovaries has been proposed as a credible biologically plausible mechanism. (Miserocchi 2008)

Among the metals detected in talcum powder products are some which are recognized carcinogens, namely nickel and chromium. It is not known how widespread was the "contamination" of talcum powder products by these metals and how high were the concentrations in the entire commercial production of talcum powder products of the past several decades, and how these exposures measure up to exposures that may cause cancer. However, evidence that asbestos and some other known carcinogens have been detected in some commercial cosmetic talcum powder products and credible mechanisms that such particles can translocate to the ovaries is an important consideration in deriving an opinion on biological plausibility, and I will consider it below in my section on biological plausibility of a causal link between talcum powder products and ovarian cancer.

Alternative formulations of baby powder include cornstarch formulations, which have become available in the past 30 years. It was possible for women to purchase and use cornstarch products or talcum powder products. Most epidemiological studies have not tried to ascertain whether the women in their studies used talc-based or cornstarch-based formulations and many women may have been unaware of the composition of the powders they used at different times. It is impossible to ascertain with certainty from most of the publications whether the reported epidemiologic results pertain to talc-based powders or cornstarch-based powders or both. Those studies that did report results for cornstarch had few women self-reporting use of cornstarch and the risk estimates were rather imprecise and unstable. For those studies that did report separately the findings for talc-based and cornstarch-based formulations, I used the results for talc-based powders. For those that did not make such distinction, I used the results combining all types of powders as reported. If it turns out that there is an increased risk associated with talc but not with cornstarch, the inability to discriminate the two in statistical analyses would have the effect of diluting the estimates of risk due to talc. That is, the RR estimates would be attenuated.

5.3.3 Routes of exposure

Some studies reported results based on particular ways of using the powders, such as on feet or perineal use or use after bathing or use on sanitary napkins or use on diaphragms or use by male partners, and so on. And many studies just reported results for all routes of perineal exposure combined. For my Main analyses, I aimed to use the reported results pertaining to all types of perineal use combined. Where the results were reported for individual routes of exposure rather than all perineal use combined, I identified the one that came closest to powdering in the perineal area from all routes.

The number of studies providing results pertaining to any of those specific routes of exposure was much less than the number providing evidence for all routes combined and insufficient to provide reliable meta-analysis results for route-specific estimates of RR. Among the route-specific reports, the one that had most often reported RR results was exposure from dusting of sanitary napkins. I will conduct a separate meta-analysis regarding the risk of ovarian cancer in relation to use of powder on sanitary napkins.

5.3.4 Questionnaire items on use of talc powders

In the case of exposure to cosmetic talc powder, the most common and realistic way of ascertaining exposure has been to question women. But there are many ways this can be done, and indeed many types of questionnaires have been used. A very simple format that has been used is to ask a question such as "have you ever used powders in your genital area?" But, the validity of the response would be enhanced if the question is framed in a more specific manner, so long as the respondent can be expected to know the answer to the more specific question. One possibility would be: "have you ever used powders that contained talc on your genital area more than once a week for at least 6 months of your life? This would include powdering your genital area directly or powdering your underwear or powdering your diaphragm or powdering your sanitary napkin." There are scores of ways such questions can be asked, and there has been variability in the methods of questioning among the different studies of powder use and ovarian cancer. In most studies, the questionnaire question about Ever Use was actually about Ever Regular Use, not Ever Occasional Use.

Among women who used powders, there can be an enormous range of usage from a few occasions in a lifetime to profuse daily usage. Among the many dimensions of talcum powder exposure that might influence the risk of cancer are the following: manner in which the talc was applied, age at which exposure began; if it ended, age at which it ended and years since it ended, frequency of use per day, week or per month, multiple applications including to genitals, undergarments, sanitary napkins, etc., and whether and how that varied at different ages. Some studies have used a single simple question, while others have used scores of questions to get at the lifetime history and many facets of powder use.

While I believe there are quality differences between the different studies in the way talc powder data have been collected, I have refrained from imposing my judgement about the quality of the questionnaire data on the selection of studies to include in meta-analyses.

5.3.5 Metrics of exposure

I used the reported results for the binary metric Ever Regular Use vs Never Regular Use, given the limitations of the available data, and using the investigators' decisions about how best to measure this. While this may seem like a simple tactic to implement, some studies were reported in such a way that in fact I had to make "judgement calls" about which of the reported results came closest to the desired metric.

For "dose-response" assessment, I used three pertinent metrics of exposure: duration (years), intensity/frequency (uses per day, week or per month), and cumulative number of applications, measured sometimes in absolute numbers or sometimes in quantiles. Of the three, the most meaningful is the cumulative number of applications.

6. My meta-analyses regarding talcum powder products and ovarian cancer: data included and results

6.1 Features of the studies

Following the exclusions indicated in Appendix Table A1, **Appendix Table A2** shows the studies that ended up being included in one or more of my meta-analyses, and brief descriptions of administrative and contextual features of each study. **Appendix Table A3** shows, for the same studies, some information about the talcum powder exposure variable and the covariates used by the authors in their control for confounding.

Appendix Table A2 shows that most studies were conducted in the USA. All but three were case-control studies and of the case-control studies, all but four used some type of population control series. Most studies had fieldwork data collection in the 1970's and 1980's; only a few studies started data collection after 2000. Table 3 shows for each study what exposure variable I was able to use to approach the notion of Ever exposed regularly to talc powder in the perineal region. Different studies had different questions in the questionnaire and different studies reported different variables. The questionnaires usually elicited lifetime use that was more than very sporadic, with terms like "regular" use. Only the Gonzalez 2016 study failed to ask about lifetime exposure before the interview; they asked about usage only in the preceding 12 months. The Gates 2010 study

asked about use of talc up to 1982 but not afterwards. Some studies asked separately about different routes of exposure and then rolled them together in statistical analyses, while some rolled all routes of exposure together in their questioning. The term that I show in Appendix Table A3 is the term that the authors reported in their publication of results; it is sometimes rather cryptic. Appendix Table A3 also shows which variables that the authors reported having used as adjustment variables. Sometimes these are variables that were explicitly included in final statistical models, and sometimes these were dealt with in a more indirect way such as a staged analysis in which a screening is conducted using a change-in-estimate procedure.

All meta-analyses were conducted using the well-known package Comprehensive Meta-Analysis Version 3. (Borenstein, M., Hedges, L., Higgins, J., & Rothstein, H. Biostat, Englewood, NJ 2013; https://www.meta-analysis.com/index.php?cart=BFZW2135997

6.2 Association between binary variable talc powdering and all types of ovarian cancer combined – data and results

6.2.1 Individual studies and results on binary exposure variable

Table 2 shows RR results, as well as the corresponding 95% confidence intervals, for each informative study included in the Main meta-analysis or in any sensitivity analyses. (I will explain this distinction below.) As I explained in Section 4.1, the single number which reflects quite well the statistical strength of a study, be it case-control or cohort, is the number of exposed cases, and I have included this parameter in Table 2. Table 2 shows the RR reported in each study by Ever (regular) use of powder in the perineal region (all routes of perineal exposure including direct powdering on genital area, on sanitary napkins, on underwear and on diaphragms). The table shows results for all types of ovarian cancer combined.

Before conducting any meta-analyses, we can peruse the results in Table 2 to observe certain patterns.

Of the 33 RR results shown in Table 2, two are below 1.0, one equals 1.0, and 30 are greater than 1.0. On the null hypothesis that there is no true association between powdering with

talc and ovarian cancer, we would expect as many of the RR estimates to be above 1.0 as to be below 1.0. The observed distribution (2 below and 30 above) is clearly and strongly in defiance of the null hypothesis. Further if we rank the RR estimates from lowest to highest, the median value, the one in the middle, would be 1.34.

This informal analysis does not take into account that the 33 estimates in Table 2 are not strictly independent of each other. There are various ways to carve out independent sets of results from this list of results in Table 2, and the meta-analyses will be designed to do that. But no matter how the studies are configured, it will be found that one or two of the RR estimates are below 1.0 and somewhere between 20 and 26 are above 1.0. Such an imbalance cannot be due to chance.

6.2.2 Strategy for Main analysis and sensitivity analyses

An investigator typically has in mind a strategy for analysing and presenting the results. There may be some judgement or assumptions involved in deciding on the strategy. The investigator may wish to see how the results would be affected if other judgements or assumptions were made. In other words, how robust are the results to alternative judgements and assumptions. Such alternative analyses are referred to as *sensitivity analyses*.

There were several dilemmas in selection of studies and results to include in the meta-analysis. I made decisions in each case that I believe provides the best basis for a meta-analysis. But in deference to other possible decisions that might have been made, I conducted some sensitivity analyses as well. I list what the dilemmas were and which options were selected for Main analyses and for sensitivity analyses.

a. Terry 2013 and Wu 2015. The Terry 2013 paper brought together data from 8 different research teams. Some of those teams had previously published their results on talc and ovarian cancer and some had not. Normally, a pooled analysis would take precedence over the individual component studies. In this case, however, there were complicating factors. The Los Angeles component study of Terry 2013 (Wu, Pike and colleagues) was conducted in stages and the Terry 2013 pooled analysis only had access to the early stage. Subsequently, Wu and colleagues carried on with their data collection, and published a

more complete set of results from their study in Wu 2015. The Terry 2013 paper contained 208 exposed cases from the Los Angeles study, whereas the Wu 2015 paper contained 701 exposed cases. In the entire Terry 2013 paper there were 2600 exposed cases. Ideally, we would wish to exclude from the 2600 exposed cases in the Terry 2013 paper, the 208 exposed cases that came from the early Los Angeles data. But that information was not available. Thus there is an 8% overlap between the exposed cases in the Terry 2013 paper and those in the Wu 2015 paper.

I adopted the following strategy. For the Main analysis, I included both Terry 2013 and Wu 2015. The 8% overlap of exposed cases is unfortunate but I believe its impact would be trivial, and in any case we will have some empirical evidence of its impact from a sensitivity analysis.

I conducted sensitivity analyses using a different strategy. The Terry 2013 paper contained a table in which the individual results of the 8 component studies were reported. I used the results as reported there for 6 of the 8 component studies, for which the Terry paper contained the latest results. For the Los Angeles study I used the result reported in Wu 2015 which was much more complete than the L.A. study result in Terry 2013. The eighth study was the study of Cramer that was one of the components of Terry 2013 but that was also reported subsequently in Cramer 2016. It is not clear whether the Cramer 2016 paper contains more up to date data than the corresponding component in Terry 2013, but it possibly does.

To summarize, the Main analysis contained pooled result from Terry 2013 and the result from Wu 2015. There were sensitivity analyses that dropped the pooled result from Terry 2013, but included the (apparent) latest published result for each of the 8 components.

b. Nurses Health Study. This cohort study was initiated in 1976 and was not a study of talcum powder products and ovarian cancer . The study involved a wide-ranging annual questionnaire which inquired about many health related issues. In 1982 there was a very succinct question about use of body powders. The cohort has been followed-up to ascertain the occurrence of cancers (or other diseases). There was a publication that contained results on talc and ovarian cancer from this study in 2000 (Gertig 2000); later, after more

years of follow-up there were two further papers presenting results on talc and ovarian cancer (Gates 2008 and Gates 2010). Clearly the Gertig result did not belong in my meta-analysis, since it was subsumed by subsequent analyses, but the choice between the Gates 2008 and Gates 2010 was not so obvious. Gates 2008 was based on a nested analysis of a subset of the cohort that probably entailed better control for confounding. Gates 2010 was based on the entire cohort and thus on a much larger sample size. The two RR estimates from the Gates papers are quite different from one another (RR=1.24 in Gates 2008 and RR=1.06 in Gates 2010). It is not clear whether the difference in results is due to the different design and analytic procedures used in the two papers. The authors did not comment on the inconsistent results.

My Main analysis included Gates 2010 but not Gates 2008. Some sensitivity analyses contained Gates 2008, but not Gates 2010.

It should be noted that whereas I did not use the Gertig paper results in the meta-analysis of Ever / Never Use of talcum powder products, I did use some dose-response results from Gertig because subsequent publications from the Nurses' Health Study did not present such results.

c. Schildkraut (2016). This was a case-control study of ovarian cancer among African American women. The fieldwork and interviewing was carried out from 2010 to 2015. The authors speculated that publicity surrounding two class action lawsuits on talc and ovarian cancer in 2014 may have subsequently induced bias in the validity of reporting of talc exposure. Consequently, in their analysis and report, they presented two sets of results, one for all women in the study, and another for those interviewed before 2014. It is impossible for me to evaluate the validity of the speculation, as it was for the authors. Consequently I will use the results from the entire sample and those from the pre-2014 sample. I refer to the entire Schildkraut study result as Schildkraut A and the pre-publicity result as Schildkraut B.

The Main analysis contained Schildkraut A. Some sensitivity analyses contained Schildkraut B.

d. Shushan (1996). This ovarian cancer case-control study, conducted in Israel, reported results on talc and ovarian cancer, but the report was quite cryptic regarding the data collection and the talc exposure variable.

The Main analysis excluded Shushan 1996. Some sensitivity analyses included Shushan 1996.

6.2.3 Results of meta-analyses on binary exposure variable for all ovarian cancers

Figure 1 shows the printout from the Comprehensive Meta-analysis (CMA) package for the Main meta-analysis for the association between ever regular use of talc powder in the genital area and all types of ovarian cancer combined. 21 RR results were used in the Main meta-analysis, but the Terry 2013 study represents 8 different study teams and 10 distinct studies. In the forest plot, I have ordered the studies in increasing magnitude of the RR estimate. It can be seen that only one study produced an RR estimate to the left of the null value of 1.0, while 19 studies produced an RR estimate to the right of the null value of 1.0.

The meta-estimate of RR is 1.28 with a 95% confidence limit from 1.19 to 1.38. The p-value is too small to register in 2 digits. This is a very highly statistically significant result. The probability of this result being attributable to chance is vanishingly small.

The 21 RR estimates in this Main meta-analysis had a fairly low p-value for heterogeneity, 0.07, but it was not statistically significant. This means that there was considerable variation in RR results across the studies, but this might have been due to chance. That there is significant variation in RR estimates is not surprising. The different studies were conducted among different populations, using different methodologies. It would be surprising if there was no variation. It is nevertheless true that in the current state of knowledge the best estimate of RR is the meta-estimate of 1.28.

Table 3 shows the results of the Main meta-analysis again and contrasts it with the results of seven sensitivity analyses that embody alternative plausible strategies for selecting studies and selecting results within studies. These alternative strategies had almost no effect. The meta-estimates of RR varied in a narrow range from 1.26 to 1.30. Even the lowest of these would lead to the conclusion that there is a highly significant association.

It can be affirmed, quite confidently, that the apparent overall elevated risk for women who had ever used such powders is not an artefact of chance variation. This conclusion is not new. It has been stated by the authors of previous meta-analyses. However, I believe this conclusion is based on the most current and reliable data now available.

From a statistical point of view, each of the studies listed in Table 2, except for one or two outliers, shows a 95% confidence interval that overlaps substantially with the confidence interval of the meta-RR estimate (1.19 – 1.38). Further, the majority of the study-specific confidence intervals (including 2 of the 3 cohort studies) include the overall meta RR of 1.28. This shows that there are few if any studies that are not compatible with the overall RR estimate.

6.2.4 Other contemporaneous meta-analyses on binary exposure variable for all ovarian cancers

I started to conduct my meta-analyses in 2015 and revised it in 2018. Towards the end of my analyses, I discovered that two other teams of researchers were carrying out meta-analyses on the same topic at almost the same time. The simultaneous and independent conduct of these three meta-analyses provides a unique opportunity to cross-validate the methodologies and results. (I knew nothing about the two others and I assume they did not know either about mine or the other meta-analysis.) It is sometimes portrayed that meta-analysis is a fairly automated procedure which should produce identical results irrespective of who carries it out. This is far from true.

Even before the statistical part of the meta-analysis is conducted, the author of a meta-analysis has to assemble all of the relevant data. That usually consists of two steps: identifying all informative studies on the topic and identifying the relevant result from each study to include in the meta-analysis. There are many ways to do these steps, and it is not surprising that different, equally competent, investigators may make different decisions about how to identify the studies and how to identify the most relevant results. This is particularly true in the area of observational epidemiology research, as opposed to clinical trials research. Research designs and methods of conduct and reporting are much more standardized in clinical trials research than they are in observational epidemiology. In the

area of research on talc and ovarian cancer (which is observational) there are many opportunities for judgement of the author of the meta-analysis to come into play, and in section 5.3.1 I have listed some of the decisions that I made, in the way I managed the selection of studies.

The two other meta-analyses were conducted by Berge et al (2018) and by Penninkilampi et al (2018). They conducted rather different search procedures than I did. Since I had already participated in the IARC review and the Langseth 2008 paper, I already had a head start on collecting the relevant scientific literature. **Appendix B** shows a 3-way comparison of the studies that were included in the meta-analyses by the three authors, and the data from each study that were judged to be most relevant by each author.

As a generalization, it can be seen that the three synchronous meta-analyses identified more or less the same studies and that in general they extracted the same result from each study; but this was not always the case. For my own meta-analysis, I was comforted to note that there was no study that was identified by one of the other meta-analyses that I had missed in my search of the literature (Appendix Table A1).

One of the main points of discordance in procedure was how the three analyses dealt with the Terry 2013 study. Namely, in my Main analysis I used the result of the pooled Ever/Never RR that was quoted by the Terry study, and dropped from consideration the various component studies of the Terry analysis. By contrast, the two others (Berge and Penninkilampi) adopted the strategy of using the results of the individual component studies rather than the overall pooled result. Berge 2018 used the results of the individual component studies as reported by Terry 2013, for most component studies, but for two component studies they used results that were reported in publications that gave results with additional cases. Penninkilampi 2018 also used individual component study results rather than the Terry 2013 pooled result. There are trade-offs between these different approaches. I prefer to use the Terry 2013 pooled result for two reasons. First, a pooled analysis with a standard set of covariates and a standard statistical model is considered superior to a meta-analysis of the components study results. Second, each publication tends to show a variety of results, and the author of the meta-analysis has to choose a

"best" one to represent the "bottom line" from each study. In the Terry pooled analysis, it was the investigators of the original studies, who were also co-authors of the pooled analysis, who chose which would be the "best" result to represent the study, and this in my opinion is more reliable than outside authors making that decision.

Table 4 shows the meta-RR results from each of the three meta-analyses. Notwithstanding the differences in choices and strategies of the three meta-analyses, the meta-RR results are quite similar, ranging from 1.22 (1.13 – 1.30) in the Berge analysis, to 1.28 (1.19 – 1.38) in my analysis, to 1.31 (1.24-1.39) in the Penninkilampi analysis. These three sets of results are really quite close to each other.

The methodology I used is sound and reliable and consistent with the high standards of my discipline. The strategy and decisions I made in relation to the studies selected and the data abstracted from each informative study is consistent with that methodology I use in my professional practice, and that has earned me recognitions and honors throughout the world.

The results shown in Table 4, are in the same "ballpark" as the meta-analysis previously conducted by Langseth 2008 and they are based on a larger pool of accumulated publications. This indicates that recent evidence is consistent with older evidence and reinforces the consistency of the evidence.

6.2.5 Meta-analysis on powdering of sanitary napkins

Tables 2-4 pertain to RRs for the combination of all routes of exposure to the perineum, including direct dusting and dusting on sanitary napkins, diaphragms, underwear, and condoms. When such an exposure variable was not provided in the paper, I used the one that came closest, with priority to dusting on the body directly. Most studies did not report RR results for every route of exposure separately. For the studies that did so, the numbers exposed were much lower than for all routes combined and there was limited statistical power in those analyses. Of the different routes, the dusting on sanitary napkins was generally the most commonly reported route apart from direct dusting. Consequently I assembled the data pertaining to dusting on sanitary napkins and conducted a meta-analysis of those results.

Table 5 shows both the individual studies that had results on sanitary napkin dusting and the meta-analysis result for those studies. The meta-RR was 1.08 (95%CI 0.89 - 1.31), heterogeneity p=0.09. Given the overlap between the confidence intervals between this meta-RR estimate for sanitary napkin powdering and the meta-RR for powdering the perineal area via any route (1.28; 95%CI 1.19 - 1.38), it cannot be affirmed that the result for sanitary napkins is statistically significantly lower than the meta-RR results in Table 3 for all routes of exposure; but the tendency is in that direction.

Berge 2018 and Penninkilampi 2018 also meta-analysed the data on use of powder on sanitary napkins. By contrast with my results, Berge 2018 reported an RR of 1.00 (95% CI 0.84-1.16) and Penninkilampi 2018 reported an RR of 1.15 (95% CI 0.94-1.41). Since their publications do not make it clear which studies and which results were used in these analyses, I cannot see easily what explains the discordance among the three meta-analyses for sanitary napkins powdering. In any case, it certainly appears that the RR was lower for application to sanitary napkins than it was for general perineal application. The interpretation of this finding is not self-evident. The different routes of exposure may entail very different frequency of exposure. For instance, whereas use of powders on sanitary napkins might involve exposure on only a few days per month, regular use on the perineal region often involves daily or near daily application. I am unaware of any evidence that would address the question of whether regular use on sanitary napkins leads to greater or lesser delivery of talc particles to the portal to the ovary than does regular powdering on the perineal region.

In any case, irrespective of the evidence regarding sanitary napkin exposure, the results in Tables 2 and 3 clearly show an association between exposure to talc in the perineal region and risk of ovarian cancer.

6.3 Dose-response – cumulative exposure, duration and frequency

An important part of the evaluation of causality is to determine whether the results display any kind of dose-response pattern. Tables 6 to 8 show results for various quantitative metrics of exposure.

Trends by cumulative exposure: **Table 6** shows results from five publications that presented results based on a cumulative amount metric. Four of the studies were based on counts of numbers of powderings, while the Cook 1997 result was based on counts of the number of days on which powdering occurred. As can be seen by perusing the column of numbers of exposed cases, the Terry 2013 results dwarf the others in terms of the statistical information they contain. The Schildkraut study, with about one-tenth as many subjects as the Terry study, nevertheless has as many subjects as the other three studies combined. The relative statistical power of the different studies is also manifested in the width of the confidence intervals.

The evaluation of the statistical significance of a trend is not a methodologically straightforward endeavour. Of particular concern is the question of whether or not the test for trend among subjects in different "dose" categories should include or exclude the unexposed category. My view is that it depends on whether or not the study results for Ever/Never exposure are part of the buffet of results presented by the authors. Namely, if the only result presented is a dose-response analysis, then it is appropriate to include the unexposed category as part of the study results. If the Ever/Never result is presented and then a dose-response analysis is conducted, it is preferable to maintain statistical independence of the two analyses by excluding the baseline unexposed category from the dose-response analyses. I will interpret the data from these studies in light of this interpretation of trend tests.

The three smallest studies in Table 6 show no evidence of a dose-response pattern. However, the estimates are so imprecise, as evidenced by the very wide confidence intervals, that they are virtually uninformative regarding the presence or the absence of dose-response.

When looking at the Terry 2013 results, which assemble data from eight teams and 10 studies, the confidence limits are much tighter and the estimates of RR much more precise. The p-value for trend (excluding the unexposed group) is 0.17. Nevertheless, with a reference value of RR=1.0 among unexposed, and with point estimates of RR in four quartiles of cumulative exposure of 1.14, 1.23. 1.22, and 1.32, these results are certainly

compatible with the presence of an underlying dose-response relationship. Note that the absence of statistical significance of the trend among the four exposed subsets is not equivalent to the demonstration of an absence of dose-response. Similarly, the Schildkraut 2016 study results, while based on only two lifetime cumulative "dose" categories with point estimates of 1.16 and 1.67, are also compatible with a dose-response pattern.

Trends by duration of exposure: **Table 7** shows the results of those studies that presented RRs by duration of use. The Terry 2013 pooled analysis did not report results by duration of use; however, some of its constituent studies did so and are included here. The numbers in each of the duration categories in each of these studies is quite small, and consequently the RR estimates are very imprecise, with wide confidence intervals. The categorisation of duration differed quite a bit among the studies and it is not easy to compare results between studies. There is no indication of a dose-response relationship in these results. Though, the wide confidence intervals make it impossible to affirm that there is evidence against dose-response. Further, the largest study showing results by duration of use, Wu 2015, did find a significant increase in risk with increasing duration.

Trends by intensity of exposure: **Table 8** shows results of those studies that reported by intensity (i.e. frequency) of usage. This ignores duration of usage. Like the results in Table 7, the results in individual studies are based on rather small numbers and they entail imprecise estimates of RR. Also like Table 7, the pattern of results is equivocal. There is no clear evidence for or against an underlying dose-response.

The Berge 2018 paper also looked at dose-response. They only looked at trends by duration of usage and frequency of usage, analogous to my Tables 7 and 8. However they actually fitted continuous variable models and found that there were significant trends in risk by duration and by frequency of exposure. They did not examine trends by cumulative exposure, and in particular they did not use the results from the Terry 2013 pooled analysis, which in my view is the most informative evidence available on dose-response.

Penninkilampi 2018 looked at risk according to long duration of usage and found no trend. They also looked at cumulative exposure with total number of applications, and they reported a slightly higher RR for women with greater than 3600 applications (RR=1.42)

compared with women who had fewer than 3600 applications (RR=1.32). I cannot determine from the paper which studies were included in this analysis and in particular whether the pooled Terry 2013 dataset was included. While the Terry 2013 and Penninkilampi 2018 papers both contained some results on dose-response, they are not included in Tables 6-8 because they are not original data collection studies; like mine, their's is a review of other studies which are contained in Tables 6-8.As indicated above, all other things being equal, the best metric of the three quantitative ones is the cumulative exposure metric, and that is the one that happens to provide the most statistically reliable data. Thus, the evidence from Table 6 overrides the weaker evidence from Tables 7 and 8. The evidence from the Terry 2013 paper is the most important piece of evidence we have on dose-response. The evidence from the Terry 2013 paper is compatible with the presence of a dose-response relationship between use of powder and ovarian cancer.

6.4 Subtypes of ovarian cancer - in particular, serous invasive tumors

Most studies that provide results on RR between talc powder and ovarian cancer provide results for all types of ovarian cancer combined. Less than half of the published studies have also provided results of the associations between talc powder exposure and various subtypes of ovarian cancer.

To the extent that talc exposure might have different effects on different subtypes of ovarian cancer, there would be a clear advantage to segregating the evidence by type of ovarian cancer and evaluating the evidence for each subtype. The serous-invasive subgroup comprises over half of all cases, and the rest are split among several other histology-behaviour subgroups (mucinous, endometroid, clear cell, others, and these can be further subdivided by invasive or borderline). Those latter subgroups entail very small numbers each and barely provide enough data, in most studies, to produce informative risk estimates. In those studies, where results were presented by histologic-behaviour subgroups, it is my judgement that there is no strong consistent pattern indicating that one subtype has higher risk than another. Of course, there is variability in point estimates of RR, but on the one hand the variability in RR estimates between ovarian cancer subtypes within studies is not greater than would be expected from chance variability (mostly, the

confidence limits overlap considerably), and on the other hand, from study to study, it is not always the same subtype that seems to have the highest or lowest relative risks.

In the largest assembly of cases subdivided by histologic subtype, the Terry 2013 pooled analysis, the results by subtype were as follows:

- Serous: n=1197; RR=1.24(1.13-1.35)
- mucinous: n=94; RR=1.06 (0.82-1.36)
- endometroid: n=304; RR=1.20 (1.03-1.40)
- clear cell: n=187; RR=1.26 (1.04-1.52).

Other than serous invasive tumors, there is no other subtype for which there are sufficient numbers of studies and sufficiently precise estimates of RR in each study to provide reliable estimates of the overall RR. While the results for endometroid and clear cell tumors show risks that are closely aligned with those for serous tumors, the result for mucinous tumors are so imprecise, because of the very small numbers of such tumors, that the estimated RR of 1.06 is very unreliable.

Consequently, and because there were multiple studies apart from Terry 2013 that presented results for serous tumors, I decided to conduct a meta-analysis for serous/invasive ovarian cancers, but not for other subgroups. The meta-analysis on serous invasive tumors will indirectly inform us also about the relative risks for other types of ovarian cancer. Namely, if the RR for serous invasive tumors is similar to that for all ovarian tumors, it will imply that the risks for other types (the complement of serous invasive tumors) will not be very different from the overall RR. If the RR for serous invasive tumors is much greater than that for all ovarian tumors, it will imply that the risks for other types (the complement of serous invasive tumors) are lower than the overall RR. Similarly, if the RR for serous invasive tumors is much lower than that for all ovarian tumors, it will

imply that the risks for other types (the complement of serous invasive tumors) are higher than that for all ovarian cancer.

Table 9 shows all the studies that reported results concerning the link between talc exposure and serous/invasive tumors. There were 8 informative studies, including Terry 2013, which carried by far the most statistical weight. The meta-RR estimate for serous/invasive tumours was 1.25 (1.1.15- 1.36). This is very similar to meta-RR for all ovarian tumors, albeit based on a smaller number of informative studies. Thus there is no persuasive evidence in these studies, taken as a whole that the effect of talc differs by histologic subtype of epithelial ovarian cancer. That is, with such a tiny difference in RRs between that for all ovarian cancers combined and that for serous invasive ovarian cancers, it can be safely inferred that the RR for other types of ovarian cancer (the complement of serous invasive) would not be far from the overall RR of 1.28.

6.5 Conclusion from meta-analyses and dose-response considerations

My opinion, based on up-to-date data and meta-analyses, is that the RR between ever perineal use of talcum powder products and ovarian cancer (all types combined) is 1.28 (95%CI 1.19-1.38). This result is highly statistically significant.

We can rule out random variability as a possible explanation for the apparent excess risks.

Further, the examination of results according to the “amount” of exposure, and notably the cumulative exposure variable used by Terry 2013, shows that the higher the exposure, the higher the risk.

Such a pattern of findings can have only two possible explanations: it must be the result of some sort of bias or confounder that operated in multiple studies or it must be the result of a real causal association.

7. Misconceptions and possible biases

In reaching my opinions, I have objectively looked at the data and scientific literature and considered the points of view of others who do not share the conclusions I have reached. There are generally two sources of disagreement: misconceptions of epidemiologic or

statistical concepts which I address below in Section 7.1 and professional judgement of the likelihood of errors and biases in the various epidemiological studies, which I address in Section 7.2.

7.1 Some prominent misconceptions in reviewing the evidence

Table 10 lists some prominent misconceptions, and I will address them here.

Misconception: "Cohort studies are more valid and informative than case-control studies."

As can be seen in Table 2, the case-control studies tended to produce higher RR estimates than the cohort studies. It has sometimes been claimed that cohort studies are more valid than case-control studies. There is no theoretical or practical reason why such a blanket assertion should be universally true. There are many factors that influence the validity of a particular result in a particular study and it cannot be reduced to any simplistic assertion that cohort studies are more valid than case-control studies, or vice versa. In the next section, I will go through a number of potential sources of distortion of results from epidemiologic studies, and I showed that some of them might occur in cohort studies, some in case-control studies, and some in both. Some of these distortions very likely occurred in some or all of these studies that provide data on talc and ovarian cancer. On balance, I believe the results of each of these case-control studies concerning female use of powders and ovarian cancer are credible, and perhaps more so (for reasons given in section 7.2), than the analogous results of each of these cohort studies.

Misconception: "Hospital-based case-control studies are more valid and informative than population-based case-control studies."

In a case-control study, the design objective is to define a study base, or a population base, in which the cases might occur, and to identify representative samples of cases and of controls in that study base. The purpose of a control group is to provide an estimate of the prevalence of exposure to the factor under study in the base population that gave rise to the cases. In many instances, the best source of controls for case-control studies is a population list of some sort. But sometimes using a population list is not feasible or

desirable, and an alternative can be to select controls from among hospital patients with conditions other than ovarian cancer. This was done in some of the ovarian cancer case-control studies.

The most common generalization made by epidemiologists is that population-based case-control studies are more valid than hospital-based case-control studies. In fact, neither this nor the opposite statement that I articulated as a Misconception, is universally correct. Validity of a case-control study depends on the specific design features and circumstances of the study.

It is possible that some types of hospital controls have patterns of usage of powders that are different from those of women in the general population, either because the powders are actually causally associated with the diseases that those women have or because their disease or condition that led them to be hospitalised induces some women to take up the use of such powders. If such a mechanism was present in a hospital-based case-control study, it would likely lead to an artificially attenuated RR, not an artificially inflated RR.

Misconception: "Counting the number of statistically significant results is a valid way of assessing consistency of results among multiple studies."

This misconception betrays a lack of understanding of statistical significance. As can be seen in Table 2, several of the individual studies listed in my meta-analysis did not find a statistically significant increase in RR. This has been cited by some as evidence that there is no real causal link.

In fact, meta-analysis is a method that was developed precisely because counting significant results is an invalid way of synthesising knowledge. Namely, a result from a single study may fail to achieve statistical significance either because there is no risk in that study, or because the statistical power of the study was limited. Meta-analysis was developed in order to combine evidence from multiple studies that may be under-powered on their own, but when combined show an effect that might be statistically significant. The meta-analysis cannot conjure a statistically significant meta-RR if the individual study RRs do not systematically lean in the direction of an excess risk, and they do so in the area of talc and ovarian cancer to a degree that cannot be explained by random fluctuation.

Misconception: "You cannot prove causality with an RR less than 2.0."

There is nothing in epidemiologic theory or practice that justifies such a statement. Indeed, this assertion about an RR $\geq$ 2.0 threshold does not exist in epidemiology. There are many well-established causal relations where the RR is less than 2.0. Table 11 lists a number of such examples. In clinical medicine also, it is very common to strive to find therapies that reduce the risk of death from some disease by as little as 10%, and several such discoveries are well documented and have been integrated in medical practice, even though the change in risk is small.

Misconception: "If a product has been used for a long time it must be safe."

It has been argued that since talc powder has been used for many decades by millions of women (and men and children), it has stood the test of time and should be considered safe. This is a specious argument.

Most agents in our environment or in our lifestyle which are now considered dangerous were used for decades or centuries without falling under a cloud of suspicion. These include such factors as cigarette smoking (many cancers and cardiovascular disease), asbestos (lung cancer), sunlight (skin cancer), ingesting very hot liquids (esophageal cancer), and many others.

Misconception: "Government agencies provide the most reliable and authoritative statements regarding the lack of carcinogenicity of talc."

Various national and international agencies have websites which list carcinogens. Examples are: National Cancer Institute (NCI), National Toxicology Program Report on Carcinogens (NTP-RoC), International Agency for Research on Cancer (IARC). It can be argued that these agencies, which undoubtedly have scientific credibility, would not put on their websites information that is out of date or invalid. However, that claim is false.

IARC has a rigorous evaluation process which is considered quite authoritative throughout the world, including in the U.S. But the evaluation is carried out at a certain point in time. The last time talc was evaluated by IARC was in 2006. Based on the evidence available then, the panel rated talc as a "possible" carcinogen. Additional evidence has been accumulated

and come to light since then, but there has not been a new evaluation by IARC. (There are potentially thousands of agents to evaluate, and IARC has resources to only evaluate a few each year. Thus they cannot keep re-evaluating the same ones as soon as new evidence is published.)

NTP-RoC is a congressionally-mandated program whereby the agency is obligated to periodically publish lists of known and suspected carcinogens. Unlike IARC, it appears that the people who make the decisions are internal RoC scientists, rather than external experts, with advice from outside experts. Also unlike IARC, the biennial reports only contain listings of those agents that have been deemed to be definite or likely carcinogens, so there does not seem to be a statutory listing of all agents that have been considered. From the minutes of a meeting of the Board of Scientific Counsellors of NTP held in 2000, it appears that the issue was deferred. I am not aware that the RoC has conducted a subsequent review of talc; although, when renominated in 2004, NTP deferred to IARC.

NCI provides a website for doctors where they indicate for each type of cancer, what are the known risk factors. Based upon my understanding, they do not carry out a rigorous evaluation along the lines of the IARC evaluations or even the NTP evaluations. It is a rather superficial process compared with the IARC process and it depends on the existing knowledge of the committee members which in a short time opines about possible associations between each of the scores of cancer types and scores of potential risk factors. This is not to argue that the members of these committees are less expert than the members of the IARC committees, but the NCI committee members have a short time (apart from their main jobs) to review hundreds of possible factor-cancer associations, whereas the IARC committee members have weeks to review just a few.

Scientists and public health agencies regularly consult the IARC evaluations and those of the NTP. The NCI website for doctors is not considered an up-to-date and cutting edge source of information. This is, of course, no reflection on the gravitas of the NCI as a whole, which has much more invested in its original research mission than in its website for doctors.

There are other organizations which may put some information about causes of cancer on their websites. Importantly, I have not seen any agency or organization, including the FDA, that conducted a rigorous evaluation of the epidemiologic and non-epidemiologic studies like we did at IARC in 2006.

Misconception: "A biological mechanism must be proven before we can establish causality"

There are innumerable examples in medical history of discoveries of risk factors or treatments that did not require knowledge of the mechanisms of pathogenesis in order to determine causality. I have compiled a few such examples from medical history and show them in **Appendix C**.

Very often, the initial suggestion was met with scepticism from the vantage point of biologic plausibility. In fact, very seldom have the essential features of biologic plausibility been worked out by the time the epidemiology has convincingly demonstrated that the association is causal. This can be asserted for the early discoveries such as the cancer causing effects of certain chemicals in dye production facilities, certain metals in various heavy industry facilities, certain emissions of combustion of fossil fuels, ionising radiation, and even cigarette smoking. In most of these examples, it was decades after the epidemiologic evidence became convincing that credible mechanistic theories were proven; though, for some, the biologic mechanisms remain unknown.

Indeed in the guidelines of the IARC Monographs, it is stated that if there is "sufficient" evidence of a risk of cancer from epidemiologic studies, then irrespective of the evidence from animal experimentation and other biologic evidence, the agent in question should be considered a Group 1 carcinogenic agent. My point here is that the demonstration of a proven biologic mechanism is not a prerequisite for demonstrating that an agent is a human carcinogen. Reliable empirical epidemiologic evidence of an association is a sufficient basis for demonstrating causality; the presence of a credible biologic mechanism enhances the degree of proof, though that often lags decades behind the general recognition of causality, as exemplified by the examples in Appendix C.

It is not my opinion that we should ignore or set aside consideration of biologic plausibility. As Hill (1965) indicated in outlining the thought process for establishing causality, biologic plausibility is one of the dimensions to be considered. But, he also cautioned that, "this is a feature I am convinced we cannot demand". Thus, as I have done in other contexts in regard to other putative carcinogens, I am able to draw causal inferences about talc irrespective of whether a causal mechanism has been proven.

Misconception: "Bradford Hill criteria comprise a checklist of necessary conditions"

As I explained in section 4.2, the "aspects" that Hill listed are not "criteria" and they are not necessary. This point has been made and is widely accepted by epidemiologists. The list of "aspects" in Hill's original paper have been rephrased and reworked in many textbooks and by most agencies that refer to them. They provide a framework and not a checklist.

7.2 Alternative explanations - Biases and errors

Before inferring that the strong statistical evidence that use of powder in the perineal area by women is associated with ovarian cancer may represent a causal relationship, I considered alternative explanations for these observations. In this section I will consider a number of potential sources of distortion of the risk estimates, under various rubrics. Some of the potential sources of distortion are unique to cohort studies, some are unique to case-control studies, and some can affect both types.

7.2.1 Bias due to non-response or non-participation

This is a potential source of bias in case-control studies.

Among all potential cases and controls who meet the study's eligibility criteria, some participate and some don't. The most common reasons for non-participation are: refusal; inability of the researchers to contact the person because they moved or are too sick or died or are otherwise unavailable; if access to the subject is via the treating physician or medical staff, there could be obstacles at that level. If the factor under study, hygiene powder use, is correlated with the likelihood of participation and if the participation rate is low, this could lead to biased estimates of RR. Such bias could artificially inflate or deflate the RR depending on how the various variables are related to each other. If response rates

are low, say below 70%, and differential both by case-control status and by exposed – non-exposed status, this could lead to biased RR estimates. For such a bias to explain the outcomes seen, it would require quite strong associations between likelihood of participation and powder use, and quite strong differences in such associations between cases and controls. In my opinion, it is very unlikely in the context of these studies that response rate differentials would be great enough to induce such large bias.

7.2.2 Recall or reporting bias

This is a potential source of bias in case-control studies.

Because the exposure history is collected retrospectively, it is subject to both non-differential recall errors (see below), and to recall or reporting bias between cases and controls. Cases and controls may have different motivation and proclivities to recall and report use of powders. If it were true that cases had a greater tendency to over-report powdering history or if controls had a greater tendency to under-report powdering, then this would lead to an artefactual exaggeration of the RR.

There are a few possible causes of such differential reporting. First, it might be hypothesized that there is a general tendency for cases in case-control studies to acknowledge behaviours or exposures with much greater frequency than controls just because they are more invested in the research than are controls. They may wrack their brains during the interview to find instances of the queried behaviour or exposure that controls don't pay much attention to during the interview, because the controls just "want to get the interview over with". If this were the case, we would systematically see elevated RRs from case-control studies for all manner of variables in all kinds of studies. But in my experience, this does not occur. (I have conducted many case-control studies, each study eliciting information on many lifestyle factors and exposures. It has not been the case that cases systematically report more exposures than controls.) Furthermore, and more pointedly, if such a phenomenon were operative in these case-control studies of ovarian cancer, we would see elevated RRs when women were questioned about the use of powders on other parts of their bodies than the perineal area. In fact, several studies did ask such questions. In the Terry 2013 analyses, based on very large numbers of women, the

overall RR for ever use of hygiene powder on non-genital areas of the body was 0.98 (0.89-1.07), in stark contrast to the analogous result for genital use of 1.24 (1.16-1.33). In other words, when questioned about powdering in non-genital areas, controls were as likely to say "yes" as cases. Clearly there was no tendency for cases to indiscriminately report exposures more frequently than controls.

A second possible reason for such a situation to arise is if there was widespread knowledge about powdering being under suspicion for ovarian cancer. In such a situation women who have heard about this might internalize the notion that powdering may have caused their cancer, and they might ruminate with such intensity on the notion that they might imagine that they had used powders at some point in the past. But for most of the period of data collection in these studies, there was very little public discussion of a possible linkage between powdering and ovarian cancer and I doubt if more than a handful of the thousands of women interviewed in these studies would have heard of such a hypothesis before being interviewed.

In my opinion recall bias is not likely to have produced the kinds of RRs we see in these studies.

7.2.3 Non-differential (or random) error in recall or reporting of exposure to powders

This is a potential source of bias that would affect both case-control and cohort studies, but not exactly in the same ways.

Reporting past history of activities and exposures is always subject to some degree of error; it can result from ambiguity or misunderstanding of the questions, failures of memory, or inattention. And this is certainly true for history of powdering. If such error is non-differential (i.e. equally present for cases and controls in the case-control context) it has an effect on RR estimates that is rather predictable. Namely, as I explained above, it has the effect of artifactually decreasing the RR. The degree of attenuation is roughly proportional to the degree of error or misclassification. If there really is a causal association between powdering and ovarian cancer, then we can be rather certain that the true RR is higher than what we can see in the various studies that have reported RRs.

Furthermore, we might make some reasonable inferences about the impact of reporting error on dose-response trends as well as on the overall RR. It is reasonable to surmise that the amount of reporting error is quite a bit higher for the details of past usage (duration of usage, intensity and frequency of past usage) than it is for the simple fact of usage. That is, there is less error in a woman's report of whether or not she ever used powders on a regular basis than in her report of the details of the usage, even if powdering behaviour may be a relatively stable habit. The consequence of this is that the RR based on ever/never usage (Table 2) is less subject to artefactual attenuation than the RRs based on categorizing the duration or intensity or cumulative amount of usage (Tables 6-8). This is a possible explanation of why there has been a much clearer signal of elevated RR for ever/never usage than there has been for dose-response.

There is likely to be more measurement error of exposure to powders in cohort studies than in case-control studies, for several reasons. First, because the cohort study questionnaires attempted to broach topics that could be relevant to many types of cancer and indeed many diseases, the questions posed in the cohort study questionnaires about talc powder use tended to be much briefer and probably less effective at eliciting valid information than the questionnaires used in case-control studies of ovarian cancer. For instance, the cohort studies did not elicit information on timing or duration of past usage, and one of the cohort studies did not even attempt to elicit information about use of talcum powder products over 12 months before the interview. Second, whereas a case-control design involves a woman looking backwards over her life from the time of incident cancer onset and thereby addressing the entire relevant period of potential exposure, the woman in a cohort study reports on her past usage as of a certain point in her life, but there may be 10-20 years subsequent in which her habits could have changed, and of which the cohort study has no knowledge. The women in the cohort studies were "locked into" their exposure category at baseline of the cohort study. If there were women who in fact started using powders after the baseline, they would be incorrectly labelled as non-users. And, if there were indeed a risk associated with use of talc powders, the risk estimate would be diluted by the incorrect inclusion of users among non-users. Accordingly, the longer such subjects are followed, the more likely such misclassification is to occur.

The age at which information is collected is a relevant consideration. In most case-control studies, the mean age of the study subjects was between 50 and 60. In the NHS cohort study the mean age at baseline questionnaire was around 40 and in the WHI it was over 60. In each study, women were asked about their past history of powder usage. Clearly the WHI women had a further stretch of time to consider than the women of the NHS and even of the case-control studies.

A particular form of measurement error may well have occurred in the Gonzalez 2016 study and produced even more attenuation of RR estimates. Namely, in their brief questionnaire on talc exposure, the question was formulated to ask women about their use of powders in the 12 months preceding the interview. While exposure to talc over the past 12 months may be correlated with exposure over the entire etiologically relevant period, which might go back decades in the life of the woman, the correlation is probably weak, and this is another source of measurement error.

7.2.4 Short follow-up periods for disease ascertainment

This is a potential source of bias that would affect cohort studies.

In a cohort study, if the period of follow-up after baseline is relatively short; and if the latency period between exposure and cancer is long, the excess risk may not be detectable because cases that would occur after long latency have not had time to occur. If this did occur, it would lead to an artificially low RR estimate.

This could have been an issue in the initial publication from the NHS, the Gertig 2000 paper. As of the Gates 2008 and Gates 2010 analyses of the NHS, the follow-up period was probably long enough and this bias should have abated. For the WHI study it was likely an issue in the Houghton 2014 paper, and it would remain so until there is much longer follow-up. It would also be an issue in the Gonzalez 2016 paper from the Sister Study, which had only 6 years of follow-up after exposure was ascertained.

7.2.5 Diagnostic error

This is a potential source of bias that would affect both case-control and cohort studies, but not exactly in the same ways.

The diagnosis of cancer is never error-free. And details of histology and staging are even more error-prone. Further, there are trends in diagnostic criteria and methods over time, as well as in the terminology and classifications used. So what we observe in these various studies of ovarian cancer represents imperfect estimates of true biologic/pathologic status. The impact of such “errors” is mainly the same as exposure measurement error, namely it would tend to artificially reduce RR estimates. Since most case-control studies start from hospital-based cancer diagnoses as the point of entry, they usually have reasonably valid diagnostic information.

In general, cohort studies tend to be more vulnerable to this source of error and bias, because disease diagnosis information is often obtained from sub-optimal sources, such as the information provided by the study subject or her family, or information obtained from death certificates. In the three cohort studies included in the meta-analysis, there were high quality verifications of diagnostic information that had been provided by the women or their families. But such verification may not be as reliable as information coming straight from hospital pathology or oncology services. I expect that this was not a major issue here, but to the extent that it did operate, it too would have led to some additional attenuation of RR estimates, as I explained above.

7.2.6 Initiation of powdering as a result of ovarian cancer

This is a potential source of bias that would affect case-control studies.

It has been speculated that women with early symptoms of ovarian cancer might take up the use of powders to help with relief of their symptoms. If so they might report that they used powders before their cancer was diagnosed and this could artificially inflate the RRs. While the women are usually questioned about the period before their cancer was diagnosed, there could be some “telescoping” so that women who start dusting after diagnosis respond in the affirmative to the questionnaire.

In the same vein, it has been speculated that treatment for ovarian cancer might produce side effects that could be relieved by powdering. And again, it might be posited that women ignore the instruction to refer the exposure question to the time before the onset of the cancer.

If the early symptoms of ovarian cancer provoke some women to start dusting the perineum to relieve some of the discomfort, or if the treatment provokes women to start dusting, this would lead to an artefactual excess RR.

I have not found any empirical evidence to support this hypothesis.

In the few datasets I have seen which describe the age distribution of initiation of powdering, there were very few patients who started in the year or two before diagnosis of the cancer. I am inclined to believe that it is virtually a non-issue, and that if it operated at all, it would only have operated on a handful of the thousands of women who were part of the various case-control studies.

7.2.7 Confounding

This is a potential source of bias that would affect both case-control and cohort studies.

If women who use powders are also more likely to be exposed to other risk factors for ovarian cancer, then it might distort the relationship between powdering and OC. The direction and the degree of distortion (bias) that would be induced depends on two components, a) the true association between the confounder and ovarian cancer, and b) the association between the confounder and dusting behaviour. Thus, depending on the direction of these two component associations, the confounding can result in artificially decreased or increased RRs. Typically, the degree of confounding is much lower than the strength of the association between the confounder and ovarian cancer. In order for a confounder to induce an artificial RR of 1.25 for dusting, it would have to have an RR much greater than 1.25 with ovarian cancer and a fairly strong correlation with dusting behaviour. Given that the main studies have controlled for the main risk factors, I consider it unlikely that this operates. Table 1 shows the covariates that were controlled for in each study, and while there is some variability between studies in the list of covariates, the main known potential confounders (age, BMI or weight, parity) have been controlled for in almost all studies. It should be noted that while smoking is a well-established risk factor for many types of cancer, it is not a risk factor for ovarian cancer; thus, there is no need to control for smoking status in studies of ovarian cancer.

A thorough and reliable investigation of potential confounders was conducted by Cramer (2016); in the large database of New England-based studies, they explored the potential confounding effect of a host of personal characteristics including demographic, reproductive, hormonal, comorbidities, activities, and exposures. None of the covariates that they explored had any meaningful confounding effect on the association between talc and ovarian cancer.

7.2.8 Publication bias

This is a potential source of bias that would affect case-control and cohort studies.

This refers to the tendency for some evidence never to "see the light of day". Namely, when results are "negative" or "null", it may be that investigators never bother to submit them for publication, or alternatively, that editors refuse to publish them. This happens, most likely, when the hypothesis under study is not particularly topical or controversial, and when the study is small. In the talc-ovarian cancer literature this would have been more likely in the pre-2000 era when there was much less scientific interest in the hypothesis linking talc to ovarian cancer. As a sensitivity analysis, I conducted a meta-analysis on the subset of studies in Table 2 that had at least 20 exposed cases. That is, I eliminated the studies from that stratum of the universe of studies that were most susceptible to publication bias. The resulting meta-RR was almost identical to those shown in Table 4. Because this has been a somewhat controversial topic in epidemiologic circles over the past 20 years, I doubt if there were large important studies with null findings on talc-ovarian cancer that went unpublished.

In their meta-analyses, Berge 2018 and Pennikilampi 2018 both showed funnel plots of the results. These are meant to detect so-called publication bias. Both of those analyses concluded that there was no publication bias.

In summary, I consider that the observed association between talc and ovarian cancer is not an artefact due to publication bias.

7.2.9 Summary comments regarding biases and errors

While the results of epidemiologic studies strongly supports the hypothesis of an association between perineal use of powders and risk of ovarian cancer, we must be wary of potential sources of error and bias that can distort an association before concluding that this association is causal. I have therefore gone through the plausible sources and types of error and bias that could potentially explain the positive association seen across the relevant studies to ascertain how likely it is that each such type was actually operative and, if so, what the nature of the impact may have been. These evaluations are based on my professional opinion as an epidemiologist having conducted, reviewed, and evaluated many hundreds if not thousands of epidemiologic studies.

Of the various types of error listed, some could artificially inflate the RR estimates and some could artificially decrease the RR estimates. Some are likely to have occurred and some are unlikely to have occurred. The one that certainly occurred and that has a non-trivial attenuating effect on RRs is random exposure misclassification (section 7.2.3). As explained above, if there is a true association, then the true RR is almost certainly greater than the estimates seen in these studies and in the resulting meta-analyses. Other types of error and bias that are highly likely to have occurred are the two that are specific to cohort studies. Namely, the Nurses' Health Study papers (Gates 2008 and Gates 2010) almost certainly suffered from an attenuated RR estimate because of the compromised reference category of "unexposed" while the Women's Health Initiative paper (Houghton 2014) and the Sister Study paper (Gonzalez 2016) almost certainly suffered from a too short follow-up period (section 7.2.4). In my opinion, the occurrence and the possible impact of other listed types of bias and error are more speculative, and less likely.

Consequently, in my opinion, the observed association between talcum powder products and ovarian cancer is unlikely to be explained by any methodological problems with the studies.

8. Bradford Hill guidelines applied to talc and ovarian cancer

The Reference Guide on Epidemiology of the Manual on Scientific Evidence (2011) states: "There is no formula or algorithm that can be used to assess whether a causal inference is

appropriate based on these guidelines." These guidelines are simply aspects that might be considered in assessing causality. I will give my assessment of how the evidence regarding talcum powder products and ovarian cancer fit into those aspects. I will use the version listed in the Reference Manual on Scientific Evidence. While there is no objective basis or scientific precedent or "scientific jurisprudence" for quantification or weighting of the various "aspects", to help the reader to understand the relevance that I attached to each "aspect" in my evaluation, I will provide an informal ranking of the importance that I attach to each aspect, in the specific context of the assessment of causality of evidence regarding talcum powder products and ovarian cancer. I will list the aspects in descending order of importance that I attach to them.

My opinions are briefly summarized in **Table 12**.

Highly important aspects in my weighting

There is a set of B-H aspects that are utterly inter-related and cannot be disassociated one from the other. In combination, they represent the most important aspect to consider in evaluation of causality of talcum powder for ovarian cancer. These include strength of association, dose-response, consideration of biases, and consistency of findings.

<u>Strength of the association</u>. This can embody both the magnitude of the RR and its statistical significance. The meta-RR estimate is 1.28. That means that the best estimate from the epidemiologic literature is that women who regularly used talcum powder products in the genital area had 28% higher risk of ovarian cancer than women who did not use such powders. As I illustrate in Table 11 with a few examples, this RR is in line with many well-recognized risk factors for cancer and other diseases. For example, it is well accepted now that people living in an urban neighborhood in which the air is highly polluted with particulate matter have between 5% and 10% excess risk of lung cancer compared with people living in a less polluted urban neighborhood. Also, it is well accepted now that workers exposed to a solvent called trichloroethylene have about a 40% higher risk of kidney cancer compared with workers not exposed to trichloroethylene. Thus, the 28% increase of ovarian cancer for women who used talcum powders is in line with many recognized risk factors. This increased risk as manifested by the meta-RR is highly

statistically significant. (Note that the statistical significance of individual studies is irrelevant to the consideration of causality; it is the totality of evidence embodied in the meta-analysis that counts.) Such a high and significant meta-RR could not have occurred by chance. This is a very important factor in how I view the evidence of causality, and it supports causality.

Dose-response relationship. If the relative risk increases when the exposure increases, it enhances the likelihood that the observed association is really causal. In studies of lifestyle habits like use of talcum powder products, the most common way is to estimate the RR in increasing categories of exposure metrics such as duration (years) of usage, or intensity of usage (frequency per day or per week or per month), or cumulative amount of usage (a combination of duration and frequency). The most sensitive of these metrics is the cumulative amount. I evaluated the published studies reported on risks according to the different metrics. By far, the most important set of results on dose-response is that from the Terry 2013 pooled analysis of 10 studies using the cumulative exposure metric. And, the next most important from a statistical weight point of view is that from Schildkraut 2016. In both of those studies, there is a clear indication of increasing risk with increasing cumulative exposure. Since the statistical power to detect a trend is less than the power to detect an overall risk, it is not surprising that the p-value for trend does not attain the conventional 0.05 level, but it remains true that these studies support a dose-response. This is an important consideration in my assessment of causality, and the evidence on dose-response that our IARC committee had available in 2006 was much less persuasive than the evidence available now.

Consideration of alternative explanations - absence of bias. There are many potential sources of bias in observational research, including in epidemiology. It is important to consider the presence of bias in each study performed or reviewed in an evaluation of causality. The possibility of bias is so multifaceted that it is impossible to reliably assign an explicit score to the likelihood of bias in a study or in a body of studies. It is also important to understand that identifying a potential source of bias is not tantamount to identifying the presence of bias. In section 7.2, I have reviewed the potential role of several types of biases and errors

that can bedevil such research. I concluded there that none of those factors would cause the apparent associations.

Consistency of findings between studies (or replication of findings). Because epidemiologic research is susceptible to errors from random variability and from different kinds of study biases, before accepting the apparent association as a generalized phenomenon, it is important to see that similar results are replicated in different studies. When these different studies also encompass different study populations in different communities, it enhances the generalizability of the inferences. Generally speaking, the observation of consistent results in different studies adds to the credibility of an inference that there really is a causal relationship. In my review of the published epidemiological studies and meta-analysis, I am impressed by the consistently elevated risk across studies. Almost all of the 30 or so studies have produced an RR greater than the null (neutral) value of 1.0. If there really were no association between talcum powder use and ovarian cancer, we would expect to see as many RRs lower than 1.0 as higher than 1.0. The pattern we see is like flipping a coin 30 times and getting a heads 28 or 29 times. The individual study RRs are not all necessarily statistically significant, but that is irrelevant because most individual studies did not have sufficient statistical power to detect RR in the range of 1.2-1.4. It is the statistical significance of the meta-RR, representing the combined evidence that has the requisite power, and that excess RR is highly statistically significant. I place great weight upon this evidence of causality and, here, believe it to be amongst the most important findings.

Moderately important aspects in my weighting

Temporal relationship. Exposure should be seen to have preceded disease. It is almost a logical truism. This is the only aspect that Bradford Hill considered to be necessary. In all of the studies I reviewed, the information elicited about talc exposure concerned the time period before cancer onset. Since it is so obviously important, the reader may wonder why I place lesser weight on this aspect. It is simply because the presence of this condition of temporality is so obvious in these studies.

Biological plausibility (coherence with existing knowledge). It is both conventional and natural to consider whether any putative association is biologically plausible. The notion of

biological plausibility is multi-facetted. In the case of talcum powder products and ovarian cancer, it can include such issues as: how such powders have been used, female anatomy and physiology, toxico-kinetics and toxicology of talc, in vitro and in vivo mechanisms of carcinogenesis, and others.

The first thing to note about this aspect that Bradford Hill listed is that it is called "biological plausibility", not "biological proof". That is, there was never any implication that a determination of causality should rest on a demonstrated proven biological mechanism. Hill was always reserved about this aspect, stating that it was not an essential prerequisite to establishing causality. As I have mentioned above, it has been common in the history of medicine and epidemiology for the elaboration of a validated biological mechanism to come much later than the discovery and demonstration of a causal association. Appendix C gives a handful of such examples but there are scores more.

Insofar as the issue of talcum powder products and ovarian cancer is concerned, there is evidence to support a few biologically plausible mechanisms. First of all, there are two possible routes that talcum powder products can take to reach the ovaries. There is published evidence that talcum powder products (and its constituents and contaminants) that are applied to the vaginal area can migrate from there to the fallopian tubes and ovaries (Venter 1979; Henderson 1986; Heller 1996) or to pelvic lymph nodes. (Cramer 2007) In addition, as has been hypothesisized and partially demonstrated in the discussion of asbestos and ovarian cancer, such particles might reach the ovaries via inhalation and translocation. (Miserocchi 2008; IARC 2012) Once the particles reach the ovaries, carcinogenesis can be triggered by the inflammation engendered by the particles. (Ness 1999; Ness 2000) There is considerable evidence that inflammation is an important mechanism for carcinogenesis (Coussens and Werb 2002; Grivennikov 2010). Alternative plausible mechanisms of carcinogenicity include talc induced oxidative stress (Buz'Zard 2007; Saed 2017; Fletcher 2018), and genotoxicity (Shukla 2009).

The evidence that commercial cosmetic talcum powder products have been shown to contain asbestos, fibrous talc, and heavy metals (Blount 1991; Paoletti 1984; Longo et al 2017, Crowley report 2018) provides a reasonable basis for hypothesizing that these

chemicals may contribute to the carcinogenicity of the talcum powder products. Asbestos is a well-known carcinogen, as are chromium and nickel compounds. It is plausible that any of these, in contact with the ovaries, can be carcinogenic.

The fact that there are credible biologically plausible mechanisms by which talcum powder products can reach the upper genital tract causing an inflammatory response, along with the presence of asbestos fibres and other carcinogens is an important consideration in support of my opinion that the genital use of talcum powder products can cause ovarian cancer.

Aspects of lesser importance in my weighting

Cessation of exposure. It is rare that there is valid evidence available to assess the impact of cessation of exposure in an observational study. In the studies on talcum powder and ovarian cancer, there is no evidence one way or the other concerning the effect of cessation of exposure. This aspect is not applicable and I place almost no weight on it.

Specificity of the association. This aspect is premised on the notion that an agent-disease association is more likely to reflect a causal association if the agent is not also associated with other diseases. In the 1960's, this seemed like a reasonable argument. In light of evidence from the past 60 years, this argument is no longer made and this aspect has fallen out of usage with the demonstration that some agents can indeed provoke multiple different pathologies. Examples include cigarette smoking, ionizing radiation and asbestos fibers.

So, I do not place much stock in this aspect. However, if I did, I would have to say that genital exposure to talc is associated with ovarian cancer and no other morbidity, which supports the 'specificity' of the relationship."

Analogy

Hill argued that if the agent is similar to another agent that has been shown to be a cause of the disease, then the agent under investigation is more likely to be a cause. The fact that exposure to asbestos fibers can cause cancers in lung, larynx mesothelial tissue and ovaries (IARC 2012) can indicate that, by analogy, talc, which is similar in some respects, might be

able to induce carcinogenesis. Thus, there is an argument for an analogy between talc and asbestos. While this aspect supports causality in Hill's framework, I consider it much less important an aspect than the ones listed above.

Coherence with other types of knowledge: Coherence with other knowledge can encompass a multitude of possibilities. This aspect is both vague and very open-ended, with no real operational instruction on how to use it. Hill gave an example in his paper, but the example was only applicable to tobacco and lung cancer. This is an aspect that, if it can be demonstrated, can enhance the likelihood of causality, but its absence cannot detract from causality. I don't consider it to have much weight in this context.

9. Contrast with IARC Monograph and other reviews

The 2006 IARC Monograph meeting, which I chaired, found that a causal relationship was "possible" between perineal talc powder exposure and ovarian cancer. I concurred with that evaluation.

It is now my professional opinion, based on the totality of the evidence that, to a reasonable degree of scientific certainty, the causal relationship between perineal talc powder exposure and ovarian cancer is "probable. "

What has changed in the years since the IARC review?

The RR estimates in Table 2 are remarkably consistent in showing a highly statistically significant excess risk. The number of published study results and scientific literature addressing the epidemiology, toxicology, molecular biology, and mechanistic studies has increased since 2006, and the evidence of excess risk has been consistently demonstrated across the past three decades.

The various possible biases that are on the table remain substantially similar to the ones that were considered by the IARC panel. At the time, we were not convinced that the apparent excess risk could be explained by those potential biases or confounding. As stated above, my review of the relevant studies and potential biases has led me to conclude that bias does not explain the consistent increased risks seen across the credible studies.

There is important new information with regard to the issue of dose-response. Contrary to the impression that the IARC panel had of a total absence of dose-response, and even a possible trend in the opposite direction, the results of three recent publications, Terry 2013 and Schildkraut 2016, using cumulative exposure metrics, and Wu 2015 using duration of exposure, all demonstrate a clear compatibility with a dose-response relationship. The recent meta-analysis of Berge 2018 supports the presence of dose-response in both duration and frequency of use. The most convincing of these is the Terry 2013 pooled analysis, which assembled a larger dataset than all other attempts to assess dose-response combined. Clearly, earlier reviews could not have integrated the results from these recent studies.

It is my opinion, based upon the above the data, there is evidence of a dose-response relationship. Penninkilampi 2018 has recently expressed a similar opinion.

10. Conclusion

The totality of evidence demonstrates that perineal or genital use of talcum powder products is associated with ovarian cancer. Based on contemporary data, my estimated RR between ever perineal use of talc powder products and ovarian cancer (all types combined) is 1.28 (95%CI 1.19-1.38). The body of epidemiologic evidence is remarkably consistent in demonstrating an excess risk. The evidence summarized in Table 6 is compatible with the presence of a dose-response relationship between cumulative exposure to talcum powder products and ovarian cancer. There are various potential sources of bias in these studies, some of which could have inflated the true RR estimate and some of which would have deflated the true RR estimate. Apart from random measurement error, which is inevitable in such studies and which tends to deflate the RR estimates, there is no certainty that the other potential biases were in fact operative and to what degree. It is my opinion, however, that neither bias nor confounding explains the consistent positive association seen across studies. Additionally, there are biologically plausible mechanisms by which talcum powder products can cause ovarian cancer.

Based on the totality of the evidence, it is my opinion, to a reasonable degree of scientific certainty, that the perineal use of talcum powder products can cause ovarian cancer. Given the seriousness of ovarian cancer and its associated morbidity, this causal risk represents an important public health issue.

11. Tables

Table 1. Steps in my evaluation of general causation between talcum powder product use and ovarian cancer

1. Identify all epidemiology study papers that present results on talc and Ovarian Cancer.
2. Extract all RR results from every paper into a database.
3. Determine which of the papers and results present truly independent relevant results.
4. Extract from each study the RR for Ever/Never use of talc in the genital area in relation to OC risk.
5. Conduct a Meta-analysis.
6. Examine the evidence about a possible dose-response relationship.
7. Consider issues of bias, confounding and other sources of error in the various studies.
8. Consider relevant opinion pieces, review articles, and agency reports.
9. Consider opinions from experts regarding possible biological mechanisms.
10. Consider all relevant aspects of association to infer causation.
11. Write report.

Table 2. Relative risk estimates between ever regular use of talcum powders products[1] in the perineal area and ovarian cancer[2], from various studies used in the Main Meta-analysis or in one or more of seven sensitivity analyses

Author	Included in Main meta-analysis	All tumours			
		Number exposed cases	RR[3]	95% CI[4]	
Booth 1989	☐	141	1.29	0.92	1.80
Chen, 1992	☐	7	3.9	0.91	10.6
Cook 1997	☐	159	1.5	1.1	2.0
Cramer 1982	☐	60	1.55	0.98	2.47
Cramer 2016		642	1.33	1.16	1.52
Gates 2008		57	1.24	0.83	1.83
Gates 2010	☐	231[5]	1.06	0.89	1.28
Godard 1998	☐	18	2.49	0.94	6.58
Gonzalez 2016	☐	17	0.73	0.44	1.2
Harlow 1989	☐	49	1.1	0.7	2.1
Harlow 1992	☐	114	1.5	1.0	2.1
Hartge 1983	☐	7	2.5	0.7	10.0

Author	Included in Main meta-analysis	All tumours			
		Number exposed cases	RR[3]	95% CI[4]	
Houghton 2014	⍰	181	1.12	0.92	1.36
Mills 2004	⍰	106	1.37	1.02	1.85
Ness 2000	⍰	161	1.5	1.1	2.0
Purdie 1995	⍰	467	1.27	1.04	1.54
Rosenblatt 1992	⍰	22	1.7	0.7	3.9
Schildkraut 2016 A[5]	⍰	248	1.44	1.11	1.86
Schildkraut 2016 B[5]		128	1.19	0.87	1.63
Shushan 1996		21	1.97	1.06	3.66
Terry 2013	⍰	2600	1.24	1.15	1.33
Terry-AUS 2013		705	1.13	0.92	1.38
Terry-DOV 2013		272	1.13	0.93	1.36
Terry-HAW 2013		74	0.99	0.70	1.41
Terry-HOP 2013		194	1.34	1.07	1.67
Terry-NCO 2013		195	1.37	1.05	1.80
Terry-NEC 2013		755	1.28	1.12	1.47
Terry-SON 2013		197	1.35	1.03	1.76

Author	Included in Main meta-analysis	All tumours			
		Number exposed cases	RR[3]	95% CI[4]	
Terry-USC 2013		208	1.36	1.06	1.74
Tzonou 1993	?	6	1.05	0.28	3.98
Whittemore 1988	?	67	1.36	0.91	2.04
Wong 1999	?	157	1.0	0.8	1.3
Wu 2015	?	701	1.46	1.27	1.69

1. In all of these studies the exposure was defined as ever use of powder in the perineal area. In most studies it was further explicitly indicated that the use was regular.
2. In this table we report the result for all types of ovarian cancer combined. With the exception of the Harlow 1989 study that was restricted to borderline tumours, we have assumed that all studies included both borderline and invasive tumours, although this was not always clear in the publications.
3. RR or OR.
4. The confidence intervals are the ones reported by the authors of the respective studies. However in its implementation procedures, the Comprehensive Meta-analysis package recomputes them to be symmetric around the point estimate, on a log scale. Consequently, in the printout of the forest plot of meta-analyses, the printed confidence interval is not always identical to the one shown in this table.
5. Estimated based on Table 1 of Gates 2010.
6. The Schildkraut 2016 case-control study presented two sets of results that both have some legitimacy for the present purpose. Schildkraut 2016A shows the results for all subjects who were interviewed in the study from 2010-2015. Schildkraut2016B shows the results for those subjects who were interviewed before 2014, and who, according to the authors, were not susceptible to having been tainted by publicity from a class action suit.

Table 3. Main meta-analysis and sensitivity analyses conducted on the association between ever regular use of talcum powder products in the perineal area and ovarian cancer (all types combined).

		RR-estimate				Heterogeneity	
Studies in meta-analysis	N*	Meta-RR	95% CI		p-value	I^2	p-value
Main Meta-Analysis - list in Figure 1 Forest plot	21	1.28	1.19	1.38	0.00	32.9	0.07
Sensitivity analyses							
Substitute Gates 2008 for Gates 2010	21	1.30	1.21	1.40	0.00	22.9	0.16
Substitute Schildkraut B for Schildkraut A	21	1.27	1.17	1.37	0.00	30.8	0.08
Add Shushan	22	1.29	1.19	1.39	0.00	33.8	0.06
Substitute List A** for Terry	27	1.27	1.19	1.35	0.00	26.2	0.10
Substitute List A for Terry and Gates 2008 for Gates 2010	27	1.29	1.21	1.37	0.00	16.6	0.22
Substitute List A for Terry and Schildkraut B for Schildkraut A	27	1.26	1.18	1.34	0.00	24.5	0.12
Substitute List A for Terry and add Shushan	28	1.28	1.20	1.36	0.00	27.4	0.09

*N: Number of RRs that went into the meta-analysis. This is not synonymous with the number of studies because some RRs (e.g. Terry 2013, Cramer 2016) embody multiple studies.

**List A studies: Cramer 2016; Wu 2015; Terry-Aus 2013; Terry-DOV 2013; Terry-Haw 2013; Terry-HOP 2013; Terry-NCO 2013; Terry SON 2013

Table 4. Comparison of results of three contemporaneous and independent meta-analyses of the association between ever regular use of talcum powder products in the perineal area and ovarian cancer.

Meta-analysis author	N*	Meta-RR	95% CI	Heterogeneity p-value
Siemiatycki 2018	21	1.28	1.19-1.38	0.07
Berge 2018	27	1.22	1.13-1.30	0.02
Penninkilompi 2018	26	1.35	1.24-1.39	0.31

* Number of published RR estimates that went into the meta-analysis. This does not necessarily correspond to the number of studies, since, for example, the Terry 2013 pooled estimate used in the Siemiatycki meta-analysis embodied 10 studies.

Table 5. Relative risk estimates between ever regular use of talcum powder products on sanitary napkins and ovarian cancer, and results of meta-analysis.

Author	**Number exposed cases**	**RR[1]**	**95% CI[2]**	
Chang 1997	51	1.26	0.81	1.96
Cook 1997	38	0.9	0.5	1.5
Cramer 1999	20	1.45	0.68	3.09
Gertig 2000	32	0.89	0.61	1.28
Harlow 1989	8	2.6	0.9	22.4[2]
Harlow 1992	9	1.1	0.4	2.8
Houghton 2014	93	0.95	0.76	1.20
Ness 2000	77	1.6	1.1	2.3
Rosenblatt 1992	21	4.8	1.3	17.8
Rosenblatt 2011	55	0.82	0.58	1.16
Whittemore 1988	5	0.62	0.21	1.80
Wong 1999	13	0.9	0.4	2.0
Meta-analysis		**1.08**	**0.89**	**1.31**
		p-value for heterogeneity = 0.09		

1. RR or OR.
2. The confidence intervals are the ones reported by the authors of the respective studies. However in its implementation procedures, the Comprehensive Meta-analysis package recomputes them to be symmetric around the point estimate, on a log scale. Consequently, in the printout of the forest plot of meta-analyses, the printed confidence interval is not always identical to the one shown in this table.

Table 6. Relative risk estimates between subgroups defined by cumulative exposure measures[1] and ovarian cancer[2], from various studies.

Author	**Cumulative applications**[3]	**Number exposed cases**	**RR**[4]	**95% C.I.**	
Cook 1997[4]	< 2000	20	1.8	0.9	3.5
	2001-5000	24	1.6	0.9	2.9
	5001-10000	21	1.2	0.6	2.4
	>10000	28	1.8	0.9	3.4
Harlow 1992	<1000	18	1.3	0.7	2.7
	1000-10000	54	1.5	0.9	2.4
	>10000	42	1.8	1.0	3.0
Mills 2004	Quartile 1	18	1.0	0.6	1.8
	Quartile 2	28	1.8	1.1	3.0
	Quartile 3	34	1.7	1.1	2.7
	Quartile 4	20	1.1	0.6	1.8
	10000+	18	0.87	0.48	1.57
Schildkraut 2016	≤3600	92	1.16	0.83	1.63
	>3600	152	1.67	1.23	2.26
Terry 2013[5]	Quartile 1	534	1.14	1.00	1.31
	Quartile 2	541	1.23	1.08	1.41
	Quartile 3	542	1.22	1.07	1.40
	Quartile 4	586	1.32	1.16	1.52

1. These were all studies that collected information on perineal use of hygiene powders in such a way as to allow construction of a cumulative measure. All of these were case control studies.
2. In this table we report the result for all types of ovarian cancer combined, as reported by the authors.
3. For the Cook study the metric was the number of days on which the woman had ever applied the powder. For the other studies the metric is based on an estimate of the total number of applications.

4. RR or OR.

5. This study was based on a pooling of studies from 8 teams. Two of the teams (Cramer 2016 and Rosenblatt 2011) published separate analyses of risk by cumulative number of applications. But these are not shown here because they are rendered redundant by the Terry 2013 pooled results.

Table 7. Relative risk estimates between subgroups defined by duration of use[1] and ovarian cancer[2], from various studies.

Author	Duration of use	Number exposed cases	RR[4]	95% C.I.	
Chang 1997	<30	60	1.7	1.1	2.6
	30-40	71	1.4	1.0	2.2
	>40	41	0.9	0.5	1.4
Cramer 1999	<20 years	55	1.9	1.2	3.0
	20-30 years	32	1.3	0.8	2.3
	>30 years	59	1.4	0.9	2.3
Cramer 2016	< 8 years of use	133	1.31	1.03	1.68
	8-19 years of use	126	1.31	1.02	1.68
	20-35 years of use	147	1.35	1.07	1.70
	>35 years of use	129	1.33	1.03	1.71
Harlow 1992	<10 years	14	1.2	0.5	2.6
	10-29 years	49	1.6	1.0	2.7
	> 30 years	51	1.6	1.0	2.7
Houghton 2014	<9 years	135	1.09	0.88	1.36
	10+ years	97	1.02	0.80	1.30
Ness 2000	<1 year	17	2.0	1.0	4.0
	1-4 years	76	1.6	1.1	2.3
	5-9 years	40	1.1	0.8	1.9
	>10 years	233	1.2	1.0	1.5
Mills 2004	<3 years	18	1.0	0.6	1.8
	4-12 years	32	1.9	1.2	3.0
	13-30 years	29	1.5	0.9	2.3
	>30 years	21	1.2	0.7	2.1

Author	Duration of use	Number exposed cases	RR[4]	95% C.I.	
Rosenblatt 2011	1-9 years	33	1.39	0.85	2.28
	10-19 years	29	1.46	0.87	2.45
	20-34 years	30	1.28	0.78	2.10
	35+ years	19	0.91	0.51	1.62
Schildkraut 2016	≤20 years	101	1.33	0.95	1.86
	>20 years	144	1.52	1.11	2.07
Whittemore 1988	1-9 years	34	1.6	1.0	2.6
	10+	50	1.1	0.7	1.7
Wong 1999	1-9 years	39	0.9	0.6	1.5
	10-19 years	49	1.4	0.9	2.2
	>20 years	101	0.9	0.6	1.2
Wu 2015	Per 5 years of exposure	1273	1.14	1.09	1.20

1. These were all studies that collected information on perineal use of hygiene powders in such a way as to allow construction of a measure of duration.
2. In this table we report the result for all types of ovarian cancer combined, as reported by the authors.
3. Years of case ascertainment or follow-up: For case-control studies this indicates the years in which cases were ascertained and data collected; for cohort studies it indicates the years of enrolment and follow-up.
4. RR or OR.

Table 8. Relative risk estimates between subgroups defined by measures of frequency of use[1] and ovarian cancer[2], from various studies.

Author	Frequency of use	Number exposed cases	RR[4]	95% C.I.	
Booth 1989	Rarely	6	0.9	0.3	2.4
	Monthly	7	0.7	0.3	1.8
	Weekly	57	2.0	1.3	3.4
	Daily	71	1.3	0.8	1.9
Chang 1997	<10 per month	76	1.8	1.2	2.7
	10-25 per month	54	1.1	0.7	1.7
	Per 10 applications per month		0.9	0.7	1.1
Cramer 1999	<30 per month	64	2.2	1.4	3.6
	30-39 per month	59	1.7	0.8	1.8
	≥40 per month	23	1.7	0.8	3.1
Cramer 2016	1-7 days per month	220	1.17	0.96	1.44
	8-29 days per month	110	1.37	1.05	1.78
	>30 days per month	205	1.46	1.20	1.78
Gates 2008	<1 per week	18	0.98	0.54	1.79
	1-6 per week	22	1.01	0.57	1.79
	Daily	35	1.44	0.88	2.37
Harlow 1992	<5 per month	32	1.5	0.8	2.7
	5-29 per month	24	1.2	0.6	2.2
	≥30 per month	58	1.8	1.1	3.0

Author	**Frequency of use**	**Number exposed cases**	**RR**[4]	**95% C.I.**	
Mills 2004	<1 per week	34	1.3	0.9	2.1
	1-3 per week	31	1.6	0.7	1.8
	4-7 per week	41	1.7	1.1	2.6
Schildkraut 2016	<Daily	88	1.12	0.80	1.58
	Daily	158	1.71	1.26	2.33
Whittemore 1988	1-20 per month	41	1.3	0.8	2.0
	>20 per month	44	1.5	0.9	2.2

1. These were all studies that collected information on perineal use of hygiene powders in such a way as to allow construction of a measure of frequency of use.
2. In this table we report the result for all types of ovarian cancer combined, as reported by the authors.
3. Years of case ascertainment or follow-up: For case-control studies this indicates the years in which cases were ascertained and data collected; for cohort studies it indicates the years of enrolment and follow-up.
4. RR or OR

Table 9. Relative risk estimates between ever regular use of talcum powder products[1] in the perineal area and invasive serous ovarian cancer, from various studies.

Author	Number exposed cases	RR[2]	95% CI[3]	
Cook 1997	71	1.7	1.1	2.5
Gates 2010	131[4]	1.06	0.84	1.35
Harlow 1992	60	1.4	0.9	2.2
Houghton 2014	105	1.13	0.84	1.51
Mills 2004	42	1.77	1.12	2.81
Schildkraut 2016	165	1.38	1.03	1.85
Terry 2013	1197	1.24	1.13	1.35
Wong 1999	136	1.2	0.7	2.1
Meta-analysis		**1.25**	**1.15**	**1.36**
			p-value for heterogeneity 0.06	

1. In all of these studies the exposure was defined as ever use of powder in the perineal area. In most studies it was further explicitly indicated that the use was regular.
2. RR or OR.
3. The confidence intervals are the ones reported by the authors of the respective studies. However in its implementation procedures, the Comprehensive Meta-analysis package recomputes them to be symmetric around the point estimate, on a log scale. Consequently, in the printout of the forest plot of meta-analyses, the printed confidence interval is not always identical to the one shown in this table.
4. Estimated based on Table 1 of Gates 2010.

Table 10. Some major misconceptions in reviewing evidence on talc and ovarian cancer

1. Cohort studies are more valid and informative than case-control studies.
2. Hospital-based case-control studies are more valid and informative than the population-based case-control studies.
3. Counting the number of “statistically significant” results is a valid way of assessing the consistency of results among multiple studies.
4. If a product has been used for a long time, it must be safe
5. You cannot prove causality with an RR less than 2.0.
6. Government agencies provide a reliable up-to-date source of scientific information.
7. A biological mechanism must be proven before we can establish causality
8. Bradford-Hill “aspects” represent a recipe list of necessary ingredients.

Table 11. Selected examples of some of the recognized causal associations that have RR less than 2.0

Agent	Disease	Approximate RR
Urban air pollution	Lung cancer	1.09[1]
Trichloroethylene	Kidney cancer	1.32[2]
Diesel engine emissions	Lung cancer	1.42[3]
Benzene	Leukemia	1.72[4]
Domestic radon gas	Lung cancer	1.29[5]
Second hand cigarette smoke	Lung cancer	1.64
Intermittent intense sun exposure	Melanoma of the skin	1.61[6]
Estrogen-progestin menopausal therapy	Breast cancer	1.59[7]

[1] Hamra GB, Guha N, Cohen A, et al (2014). Outdoor Particulate Matter Exposure and Lung Cancer: A Systematic Review and Meta-Analysis, *Environ Health Perspect* 122:906-911.
[2] Karami S, Lan Q, Rothman N, et al (2012). Occupational trichloroethylene exposure and kidney cancer risk: a meta-analysis. *Occupational and Environmental Medicine* 69:858-867.
[3] Mahjub H, Sadri G (2006). Meta-analysis of case-referent studies of specific environmental or occupational pollutants on lung cancer. *Indian Journal of Cancer* 43(4):169-173.
[4] Khalade A, Jaakkola MS, Pukkala E, Jaakkola JJ (2010). Exposure to benzene at work and the risk of leukemia: a systematic review and meta-analysis. *Environmental Health* 9(31):1-8.
[5] Zhang Z-L, Sun J, Dong J-Y, et al (2012). Residential Radon and Lung Cancer Risk: An Updated Meta-analysis of Case-control Studies. *Asian Pac J Cancer Prev* 13:2459-2465.
[6] Gandini S, Sera F, Cattaruzza MS, et al (2004). Meta-analysis of risk factors for cutaneous melanoma: II. Sun exposure. *European Journal of Cancer* 41:45-60.
[7] Kim S, Ko Y, Lee HJ, Lim J (2018). Menopausal hormone therapy and the risk of breast cancer by histological type and race: a meta-analysis of randomized controlled trials and cohort studies. *Breast Cancer Research and Treatment* 170(3):667-675.

Cigarette smoking	Cardiovascular disease	1.6[8]
Physically inactive (compared with physically active)[9]	Hypertension	1.19
	Diabetes	1.12
Low fruit and vegetable diet	Cardiovascular disease	1.09[10]

[8] Doll R, Peto R, Boreham J, Sutherland I (2004). Mortality in relation to smoking: 50 years' observations on British male doctors, *British Medical Journal*, 328(7455):1519.

[9] Carnethon MR, Gidding SS, Nehgme R, Sidney S, Jacobs, Jr DR, Liu K (2003). Cardiorespiratory Fitness in Young Adulthood and the Development of Cardiovascular Disease Risk Factors. *JAMA*, 290(23):3092–3100

[10] Aune D, Giovannucci E, Boffetta P, Fadnes L, Keum N, Norat T, Greenwood D, Riboli E, Vatten L, Tonstad S (2017). Fruit and vegetable intake and the risk of cardiovascular disease, total cancer and all-cause mortality – a systematic review and dose-response meta-analysis of prospective studies, International Journal of Epidemiology 43(3):1029-1056. (This RR estimate is computed from the reciprocal of the High fruit and vegetable variable that was reported by the authors. That is, 1/0.92).

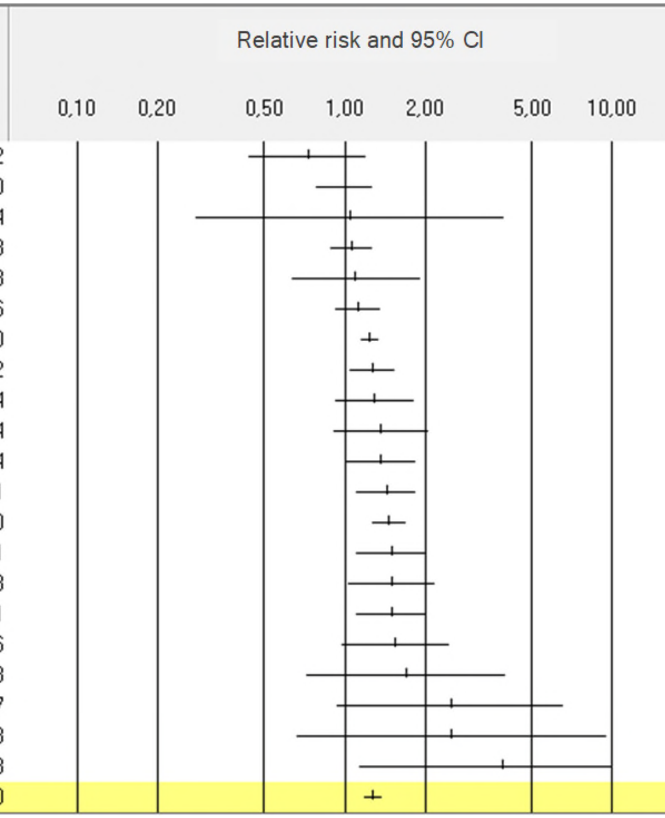
Table 12. Bradford Hill aspects in relation to perineal talc exposure and ovarian cancer

Aspect	Brief comment	Weight in evaluating causality
Strength of the association	There are stronger associations and there are weaker associations	High
Dose response relationship	Reasonably clear increase in risk with increasing exposure	High
Consideration of alternative explanations – absence of bias	Yes considered, and none is compelling	High
Replication of the findings	Very strong, almost all studies support association	High
Temporal relationship	Exposure preceded disease in all studies	Moderate
Biological plausibility	There are plausible mechanisms	Moderate
Cessation of exposure	Not applicable.	Less
Specificity of the association	Yes, talc is not associated with a multitude of diseases	Less
Coherence with other knowledge	Could be similar to asbestos carcinogenicity	Less
Analogy		Less

12. Figures

Figure 1. Meta-analysis of relative risk of ovarian cancer (all types combined) among women who regularly used talc powder in the perineal area, based on all informative studies, studies ordered by magnitude of RR.

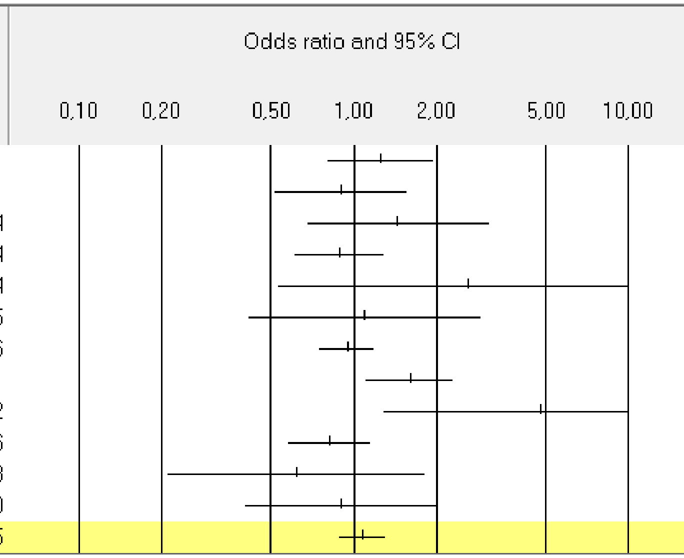
Model	Study name	Statistics for each study				Weight (Random)
		Risk ratio	Lower limit	Upper limit	p-Value	Relative weight
	Gonzalez 2016	0,73	0,44	1,21	0,22	2,05
	Wong 1999	1,00	0,78	1,27	1,00	6,50
	Tzonou 1993	1,05	0,28	3,96	0,94	0,32
	Gates 2010	1,06	0,88	1,27	0,53	9,17
	Harlow 1989	1,10	0,64	1,91	0,73	1,74
	Houghton 2014	1,12	0,92	1,36	0,26	8,48
	Terry 2013	1,24	1,15	1,33	0,00	16,37
	Purdie 1995	1,27	1,04	1,55	0,02	8,43
	Booth 1989	1,29	0,92	1,80	0,14	4,05
	Whittemore 1988	1,36	0,91	2,04	0,14	3,00
	Mills 2004	1,37	1,02	1,85	0,04	4,87
	Schildkraut 2016 A	1,44	1,11	1,86	0,01	5,98
	Wu 2015	1,46	1,27	1,68	0,00	11,46
	Cook 1997	1,50	1,11	2,02	0,01	4,84
	Harlow 1992	1,50	1,04	2,17	0,03	3,45
	Ness 2000	1,50	1,11	2,02	0,01	4,84
	Cramer 1982	1,55	0,98	2,46	0,06	2,37
	Rosenblatt 1992	1,70	0,72	4,01	0,23	0,75
	Godard 1998	2,49	0,94	6,59	0,07	0,59
	Hartge 1983	2,50	0,66	9,45	0,18	0,32
	Chen, 1992	3,90	1,14	13,31	0,03	0,38
Random		1,28	1,19	1,38	0,00	



Model		Effect size and 95% interval			Test of null (2-Tail)		Heterogeneity				Tau-squared			
Model	Number Studies	Point estimate	Lower limit	Upper limit	Z-value	P-value	Q-value	df (Q)	P-value	I-squared	Tau Squared	Standard Error	Variance	Tau
Fixed	21	1,264	1,204	1,327	9,474	0,000	29,813	20	0,073	32,916	0,008	0,008	0,000	0,088
Random effects	21	1,280	1,186	1,381	6,364	0,000								

Figure 2. Meta-analysis of relative risk of ovarian cancer (all types combined) among women who regularly used talcum powder products on sanitary napkins, based on all informative studies.

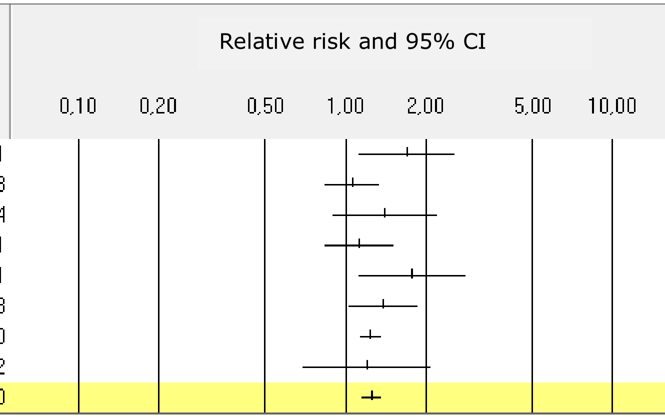
Model	Study name	Statistics for each study				Weight (Random)
		Odds ratio	Lower limit	Upper limit	p-Value	Relative weight
	Chang 1997	1,26	0,81	1,96	0,31	11,14
	Cook 1997	0,90	0,52	1,56	0,71	8,47
	Cramer 1999	1,45	0,68	3,09	0,34	5,26
	Gertig 2000	0,89	0,61	1,29	0,54	13,44
	Harlow 1989	2,60	0,53	12,74	0,24	1,41
	Harlow 1992	1,10	0,42	2,91	0,85	3,45
	Houghton 2014	0,95	0,76	1,19	0,66	19,32
	Ness 2000	1,60	1,11	2,31	0,01	13,50
	Rosenblatt 1992	4,80	1,30	17,76	0,02	2,03
	Rosenblatt 2011	0,82	0,58	1,16	0,26	14,32
	Whittemore 1988	0,62	0,21	1,82	0,38	2,90
	Wong 1999	0,90	0,40	2,01	0,80	4,76
Random		1,08	0,89	1,31	0,45	



Model	Number Studies	Point estimate	Lower limit	Upper limit	Z-value	P-value	Q-value	df (Q)	P-value	I-squared	Tau Squared	Standard Error	Variance	Tau
		Effect size and 95% interval			Test of null (2-Tail)		Heterogeneity				Tau-squared			
Fixed	12	1,041	0,911	1,189	0,591	0,554	17,614	11	0,091	37,551	0,037	0,045	0,002	0,193
Random effects	12	1,078	0,888	1,309	0,763	0,445								

Figure 3. Meta-analysis of relative risk of invasive serous ovarian cancer among women who regularly used talcum powder products in the perineal area, based on all informative studies

Model	Study name	Statistics for each study				Weight (Random)
		Risk ratio	Lower limit	Upper limit	p-Value	Relative weight
	Cook 1997	1,70	1,13	2,56	0,01	4,13
	Gates 2010	1,06	0,84	1,34	0,63	11,79
	Harlow 1992	1,40	0,90	2,19	0,14	3,49
	Houghton 2014	1,13	0,84	1,52	0,41	7,90
	Mills 2004	1,77	1,12	2,80	0,01	3,30
	Schildkraut 2016	1,38	1,03	1,85	0,03	7,92
	Terry 2013	1,24	1,13	1,36	0,00	59,13
	Wong 1999	1,20	0,69	2,08	0,52	2,33
Random		1,25	1,15	1,36	0,00	



Model		Effect size and 95% interval			Test of null (2-Tail)		Heterogeneity				Tau-squared			
Model	Number Studies	Point estimate	Lower limit	Upper limit	Z-value	P-value	Q-value	df (Q)	P-value	I-squared	Tau Squared	Standard Error	Variance	Tau
Fixed	8	1,250	1,161	1,345	5,963	0,000	7,401	7	0,388	5,422	0,001	0,011	0,000	0,033
Random effects	8	1,254	1,152	1,364	5,249	0,000								

13. Appendix A

Appendix Table A1. Papers that contain some results on the association between exposure to perineal talc and ovarian cancer, and whether the paper was included in my meta-analyses of the binary Ever/Never exposed variable

Author	Included/excluded	Reasons for exclusion
Booth 1989	Core Inclusion	
Chang 1997	Core Inclusion	
Chen 1992	Core Inclusion	
Cook 1997	Core Inclusion	
Cramer 1982	Core Inclusion	
Cramer 1995	Excluded	Included in Terry 2013 and in Cramer 2016
Cramer 1999	Excluded	Included in Terry 2013 and in Cramer 2016
Cramer 2005	Excluded	Included in Terry 2013 and in Cramer 2016
Cramer 2016	Excluded when Terry 2013 is included	Considerable overlap between this and the Terry 2013 NEC component
Eltabbakh 1998	Excluded	Cases were peritoneal cancer and controls were ovarian cancer
Gates 2008[2-]	Included in one sensitivity analysis	Overlap with Gates 2010

Author	Included/excluded	Reasons for exclusion
Gates 2010[2]	Included in all analyses except one sensitivity analysis	This may be a more complete analysis than Gates 2008, but the degree of overlap is unclear.
Gertig 2000	Excluded	Subsumed in Gates 2008 and Gates 2010
Godard 1998	Core inclusion	
Gonzalez 2016	Core inclusion	
Green 1997	Excluded	This appears to be an analysis of a subset of the subjects in Purdie 1995
Hankinson 1993	Excluded	Numerical results were not presented.
Harlow 1989	Core inclusion	
Harlow 1992	Core inclusion	
Hartge 1983	Core inclusion	
Houghton 2014	Core inclusion	
Jordan 2007	Excluded	Benign tumours only
Kurta 2012	Excluded	Included in Terry 2013
Langseth 2004	Excluded	Not based on perineal application of cosmetic powder.
Lo-Ciganic 2012	Excluded	Same study as Kurta 2012 and included in Terry 2013.
Merrit 2008	Excluded	Included in Terry 2013

Author	Included/excluded	Reasons for exclusion
Mills 2004	Core inclusion	
Moorman 2009	Excluded	Included in Terry 2013
Pike 2004	Excluded	Included in Terry 2013
Purdie 1995	Core inclusion	
Ness 2000	Core inclusion	
Rosenblatt 1992	Core inclusion	
Rosenblatt 2011	Core inclusion	
Schildkraut 2016	Core inclusion	
Shushan 1996	Included in sensitivity analysis	Unclear on how they obtained data on talc exposure or what the route of exposure was
Terry 2013	Included in Main analysis, but replaced by component studies in sensitivity analyses	
Tzonou 1983	Core inclusion	
Whittemore 1988	Core inclusion	
Wong 1999	Core inclusion	
Wu 2015	Core inclusion	

Appendix Table A2. Some administrative and contextual information on the studies used in the following tables

Author	Study location	Years of case ascertainment/ follow-up[1]	Type of study
Booth 1989	London, Oxford UK	1978-1983	Case-control; Hospital controls
Chen 1992	Beijing Cancer Registry	1984-1986	Case-control; Population controls
Cook 1997	Washington State	1986-1988	Case-control; Population controls
Cramer 1982	Boston	1978-1981	Case-control; Population controls
Cramer 2016	New England	1992-2008	Case-control; Population controls
Gates 2008[2-]	USA – NHS study	1976-2004	Case-control nested in Cohort (US nurses)
Gates 2010[2]	USA – pooled 2 cohorts of nurses NHS and NHSII	1976-2004 1989-2005	Cohort (US Nurses)
Godard 1998	Montreal, Canada	1995-1996	Case-control; Population controls
Gonzalez 2016	Puerto Rico and 11 States USA	2003-2014	Cohort
Harlow 1989	Washington State	1980-1985	Case-control; Population controls
Harlow 1992	Boston	1984-1987	Case-control; Population controls

Author	Study location	Years of case ascertainment/ follow-up[1]	Type of study
Hartge 1983	Washington, DC	1974-77	Case-control; Population controls
Houghton 2014	USA	1993-2012	Cohort (WHI)
Mills 2004	California	2000-2001	Case-control; Population controls
Ness 2000	Pennsylvania, New Jersey, Delaware	1994-1998	Case-control; Population controls
Purdie 1995	Australia	1990-1993	Case-control; Population controls
Rosenblatt 1992	Baltimore	1981-1985	Case-control; Hospital controls
Schildkraut 2016	USA	2010-2015	Case-control; Population controls
Shushan 1996	Israel	1990-1993	Case-control Population controls
Terry 2013	Pooled 8 studies: USA & Australia	1984-2008	Case-control; Population controls
Terry-AUS 2013	Australia	2002-2006	Case-control Population controls
Terry – DOV[3] 2013	Washington State	2002-2009	Case-control Population controls
Terry – HAW 2013	Hawaii	1993-2008	Case-control Population controls

Author	Study location	Years of case ascertainment/ follow-up[1]	Type of study
Terry – HOP 2013	Pennsylvania, Ohio, Western NY State	2003-2008	Case-control Population controls
Terry – NCO 2013	North Carolina	1999-2008	Case-control Population controls
Terry – NEC 2013	Massachusetts, New Hampshire	1992-2006	Case-control Population controls
Terry – SON 2013	Southern Ontario	1989-1992	Case-control Population controls
Terry – USC 2013	Los Angeles County	1992-1998	Case-control Population controls
Tzonou 1983	Athens	1989-1991	Case-control; Controls – hospital visitors
Whittemore 1988	San Francisco	1983-1985	Case-control; Hospital & population controls
Wong 1999	Buffalo	1982-1992	Case-control; Hospital controls
Wu 2015	Los Angeles County	1992-2008	Case-control; Population controls

1. Years of case ascertainment or follow-up: For case-control studies this indicates the years in which cases were ascertained and data collected; for cohort studies it indicates the years of enrolment and follow-up.
2. The Gates 2008 and Gates 2010 papers are both derived from the U.S. Nurses Cohort. The latter represent results for all cases diagnosed up to 2006 and analysed in the cohort framework. The former represents results for a sub-set of cases that were selected for a nested c-c analysis. The number of exposed cases was not given in the Gates 2010 paper.
3. Terry – DOV 2013: the information in Terry 2013 is updated information included in Rosenblatt 2011.

Appendix Table A3. Covariates used in the analyses and exposure variables in the studies used in the following tables.

Author	**Exposure variable selected**	**Covariates used in analysis**
Booth 1989	At least monthly use	Since the authors did not present results for “ever” exposed, I calculated the OR from crude numbers in their tables. Therefore the OR presented is a crude one. However, results presented in Table 7 adjusted for age and social class
Chen 1992	Dusting perineum or lower abdomen > 3 months	Education
Cook 1997	Lifetime perineal application	Age
Cramer 1982	Any use as dusting powder and/or on sanitary napkins	Parity; menopausal status
Cramer 2016	Any talc use	Age; study center (MA, NH); BMI; primary relative with breast or ovarian cancer; parity; OC use; tubal ligation
Gates 2008[1-]	Regular genital talc use (1 per week or more)	Age; OC[2] use; parity; BMI; post-menopausal hormone use
Gates 2010[1]	Regular genital talc use (1 per week or more)	Age; BMI; physical activity; smoking; family history of breast or ovarian ca; OC use; tubal ligation; hysterectomy; age menopause; estrogen use
Godard 1998	Ever use of talc on perineum	Age; reproductive factors; OC use; tubal ligation; alcohol use; breast and abdominal surgery
Gonzalez 2016	Talc use in the past 12 months	Race; body mass index; parity; duration of oral contraceptive use; baseline menopause status; and patency

Author	Exposure variable selected	Covariates used in analysis
Harlow 1989	Any genital talc use	Age; county; parity; OC use
Harlow 1992	Any genital talc use	Age; county; parity; marital status; education; religion; weight; use of sanitary napkins; douching
Hartge 1983	Any genital talc use	Race; age; gravidity
Houghton 2014	Combined use: longest duration of use among the applications to genitals, sanitary napkins and diaphragms	Age; race; OC use; HRT[3] use; family history of ovarian ca; age at last birth; BMI; smoking; tubal ligation; parity
Mills 2004	Ever use of talcum powder in genital area	Age; race/ethnicity; OC use; breast-feeding
Ness 2000	Genital rectal talc use	Age; parity; family history of ovarian ca;
Purdie 1995	Ever used talc in perineal region	Age; parity; duration of OC use; education; BMI; smoking; family history of ovarian ca
Rosenblatt 1992	Ever use of bath talc	Number of live births
Schildkraut 2016	Regular use of talc, cornstarch, baby or deodorising powder – at least once a month for 6 months	Age at diagnosis/interview; study site; education; tubal ligation; parity; BMI duration of OC use first degree family history of breast or ovarian cancer; and interview year
Shushan 1996	Talc use – never, seldom, moderate, a lot	Crude OR

Author	Exposure variable selected	Covariates used in analysis
Terry 2013 – all components of the pooled analysis	Genital powder use	Age; OC use; parity; BMI; tubal ligation; ethnicity; race; tubal ligation; hysterectomy; breastfeeding
Tzonou 1983	Ever use of talc in perineal region	Age; years of schooling; weight before onset of the disease; age at menarche; menopausal status and age at menopause; parity and age at first birth; tobacco smoking; coffee drinking; consumption of alcoholic beverages; hair dyeing; use of analgesics and tranquilizers/hypnotics
Whittemore 1988	Talcum powder used on any two of perineum, sanitary pads and diaphragm	Age; race; hospital; parity
Wong 1999	Ever use of talc on genital region or thighs	Age; income; education; geographic location; OC use; smoking; family history of ovarian ca; age at menarche; menopausal status; tubal ligation or hysterectomy
Wu 2015	Genital talc use >1 year	Age; race/ethnicity; interviewer; reproductive variables; sociodemographic variables; medical history; hormonal variables; BMI.

1. The Gates 2008 and Gates 2010 papers are both derived from the U.S. Nurses Cohort. The latter represent results for all cases diagnosed up to 2006 and analysed in the cohort framework. The former represents results for a sub-set of cases that were selected for a nested c-c analysis. The number of exposed cases was not given in the Gates 2010 paper.
2. OC: oral contraceptive
3. HRT: hormone replacement therapy

14. Appendix B

Comparison of studies used and results extracted from articles referenced in three different meta-analyses.*

Penninkilampi 2018 **Study / RR(95%CI)**	**Berge 2018** **Study / RR(95%CI)**	**Siemiatycki 2018** **Study / RR(95%CI)**
Booth 1989 1.30 (0.94-1.80)	***Booth 1989*** ***1.29 (0.92 - 1.80)***	***Booth 1989*** ***1.29 (0.92 - 1.80)***
Chang 1997 1.42 (1.08 – 1.86)	Chang 1997 1.35 (1.03 - 1.76)	
Chen, 1992 3.90 (1.43 – 10.60)	***Chen, 1992*** ***3.90 (0.91 - 10.60)***	***Chen, 1992*** ***3.90 (0.91 - 10.60)***
Cook 1997 ***1.50 (1.11 – 2.02)***	***Cook 1997*** ***1.50 (1.10 - 2.00)***	***Cook 1997*** ***1.50 (1.10 - 2.00)***
Cramer 1982 1.60 (1.21 – 2.12)	***Cramer 1982*** ***1.92 (1.27 - 2.89)***	***Cramer 1982*** ***1.92 (1.27 - 2.89)***
Cramer 2016 1.42 (1.03 – 1.95	Cramer 2016 1.32 (1.14 - 1.50)	Cramer 2016 1.33 (1.16 – 1.52)
		Gates 2008 1.24 (0.83 - 1.83)
	Gates 2010 ***1.06 (0.89 - 1.28)***	***Gates 2010*** ***1.06 (0.89 - 1.28)***
Gertig 2000 1.09 (0.86 – 1.38)		

Penninkilampi 2018 Study / RR(95%CI)	Berge 2018 Study / RR(95%CI)	Siemiatycki 2018 Study / RR(95%CI)
Godard 1998 ***2.49 (0.94 - 6.58)***	***Godard 1998*** ***2.49 (0.94 - 6.58)***	***Godard 1998*** ***2.49 (0.94 - 6.58)***
Gonzalez 2016 ***0.73 (0.44 - 1.20)***	***Gonzalez 2016*** ***0.73 (0.44 - 1.20)***	***Gonzalez 2016*** ***0.73 (0.44 - 1.20)***
	Goodman 2008 0.99 (0.7 - 1.41)	
Green 1997 1.30 (1.06 – 1.60)		
Harlow 1989 1.10 (0.58 – 2.10)	***Harlow 1989*** ***1.10 (0.70 - 2.10)***	***Harlow 1989*** ***1.10 (0.70 – 2.10)***
	Harlow 1992 ***1.50 (1.00 - 2.10)***	***Harlow 1992*** ***1.50 (1.00 – 2.10)***
Hartge 1983 ***2.50 (0.66 – 9.45)***	***Hartge 1983*** ***2.50 (0.70 - 10.00)***	Hartge 1983 0.70 (0.40 - 1.10)
Houghton 2014 ***1.12 (0.92 – 1.36)***	Houghton 2014 1.06 (0.87 - 1.28)	***Houghton 2014*** ***1.12 (0.92 – 1.36)***
Kurta 2012 1.40 (1.16 – 1.69)		
	Lo-Ciganic 2012 1.34 (1.07 - 1.66)	

Penninkilampi 2018 **Study / RR(95%CI)**	**Berge 2018** **Study / RR(95%CI)**	**Siemiatycki 2018** **Study / RR(95%CI)**
Merritt 2008 1.17 (1.01 – 1.36)	Merritt 2008 1.13 (0.92 - 1.38)	
Mills 2004 ***1.37 (1.02 - 1.85)***	***Mills 2004*** ***1.37 (1.02 - 1.85)***	***Mills 2004*** ***1.37 (1.02 - 1.85)***
	Moorman 2009 1.37 (1.05 - 1.8)	
Ness 2000 ***1.50 (1.10 - 2.02)***	***Ness 2000*** ***1.50 (1.10 - 2.00)***	***Ness 2000*** ***1.50 (1.10 - 2.00)***
Purdie 1995 ***1.27 (1.04 - 1.54)***	***Purdie 1995*** ***1.27 (1.04 - 1.54)***	***Purdie 1995*** ***1.27 (1.04 - 1.54)***
Rosenblatt 1992 1.70 (0.72 – 4.01)	***Rosenblatt 1992*** ***1.70 (0.70 - 3.90)***	***Rosenblatt 1992*** ***1.70 (0.70 - 3.90)***
Rosenblatt 2011 1.27 (0.97 – 1.66)	Rosenblatt 2011 1.13 (0.93 - 1.36)	
Schildkraut 2016 ***1.44 (1.11 - 1.86)***	***Schildkraut 2016*** ***1.44 (1.11 - 1.86)***	***Schildkraut 2016 A*** ***1.44 (1.11 - 1.86)***
		Schildkraut 2016 B 1.19 (0.87 - 1.63)
Shushan 1996 2.00 (1.11 – 3.60)		Shushan 1996 1.97 (1.06 – 3.66)

Penninkilampi 2018 **Study / RR(95%CI)**	**Berge 2018** **Study / RR(95%CI)**	**Siemiatycki 2018** **Study / RR(95%CI)**
		Terry 2013 1.24 (1.15 - 1.33)
Tzonou 1993 ***1.05 (0.28 - 3.96)***	***Tzonou 1993*** ***1.05 (0.28 - 3.98)***	***Tzonou 1993*** ***1.05 (0.28 - 3.98)***
Whittemore 1988 1.40 (0.98 – 2.00)	***Whittemore 1988*** ***1.36 (0.91 - 2.04)***	***Whittemore 1988*** ***1.36 (0.91 - 2.04)***
Wong 1999 0.92 (0.24 – 3.57)	***Wong 1999*** ***1.00 (0.80 - 1.30)***	***Wong 1999*** ***1.00 (0.80 - 1.30)***
Wu 2015 1.32 (1.14 – 1.52)	***Wu 2015*** ***1.46 (1.27 - 1.69)***	***Wu 2015*** ***1.46 (1.27 - 1.69)***

* When two or three of the meta-analyses extracted the identical results from the source paper, it is indicated with italic characters.

15. Appendix C

Examples of historic discoveries made on the basis of empirical observation of an association, without the existence of a validated biological mechanism of action.

- Jenner (18th century) discovered that smallpox could be prevented by "vaccinating" people. This was based on observation of the effect of exposure to cowpox. He had no idea about viruses or the biology of smallpox. He only knew that the "association" he observed between vaccination and the prevention of smallpox was so strong as to convince him it was causal. Millions of lives were saved as a result.
- Snow (19th century) discovered that cholera was caused by something in the water supply. He did not know what the pathogen was or how it produced the disease, but he showed with sufficient epidemiologic proof that drinking water from a polluted source produced much higher rates than drinking water from a clean source. Despite the ignorance of biological mechanisms, the public health authorities acted on his findings and thereby greatly reduced the incidence of cholera.
- Rheumatic fever and rheumatic heart disease were quite common causes of disease and death, striking relatively young people. For many decades it was recognized that there was an association between infection with the streptococcus bacterium and rheumatic heart disease, but it was not understood how the bacterium could have such an effect. The lack of understanding of the biological mechanisms did not get in the way of prevention of rheumatic heart disease by preventing and treating streptococcus infection.
- In the 1930's and 1940's, it was noticed that communities with high natural levels of fluoride in the water had much lower levels of dental caries than communities with low fluoride levels. Additional observational research confirmed the clear causal relationship and this led to extensive use of fluoride in various ways to reduce dental disease. But, all this occurred

before the mechanisms by which fluoride acted on teeth were understood. And, indeed the mechanisms are still not fully understood.

- In the late 1940's and early 1950's, evidence was accumulating that cigarette smokers had higher rates of lung cancer than non-smokers. This "association" was ridiculed at the time, among other reasons, because there was no proven biological mechanism. Attempts to replicate smoking-related lung cancer incidence in laboratory animals were largely unsuccessful. Nor was there a deep understanding of the cellular processes that allow the inhalation of cigarette smoke to culminate in a tumor. Scores of studies later and many decades later, the outlines of a credible biological mechanism began to emerge. The absence of a proven biological mechanism did not hinder the US Surgeon General and other national bodies from concluding that there was a causal link as early as the 1960's.
- Many chemicals have been found to be carcinogenic as a result of epidemiologic studies among workers. Examples of these are asbestos, silica, nickel compounds, chromium compounds, benzene, and others. Some of these discoveries go back to the first half of the 20th century, and, for most of them, many decades passed between the time they were recognized as carcinogens, on the basis of epidemiologic associations, and the elaboration of credible mechanisms of how they induce cancer. (Siemiatycki 2015) Most known carcinogens were first discovered empirically by medical doctors or epidemiologists, usually as part of large data collection activities or just plain astute observation on the part of medical doctors.

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JNJAZ55_000015127-286

JNJMX68_000003728-29

JNJMX68_000004646-47

JNJMX68_000004996-5044

JNJMX68_000006792

JNJMX68_000008982-9004

JNJMX68_000011720-25

JNJMX68_000012858

JNJMX68_000012745-49

JNJMX68_000013019-20

JNJMX68_000013464-66

JNJMX68_000017401-43

JNJMX68_000019698-99

JNJN000294462

JNJNL61_000000134-36

JNJNL61_000002666-73

JNJNL61_000006431-32

JNJNL61_000008084-89

JNJNL61_000014431-37

JNJNL61_000020359

JNJNL61_000029410-36

JNJNL61_000052427

JNJNL61_000061857

JNJNL61_000063473

JNJNL61_000064366-67

JNJNL61_000079334-35

JNJTALC000716827-45

LUZ000250

LUZ000566-67

LUZ001017-22

LUZ001298-1303

LUZ001326-27

LUZ001441-44

LUZ001719-20

LUZ001873-76

LUZ002733-51

LUZ003202-03

LUZ003204

LUZ003264-67

LUZ004656-65

LUZ005090-91

LUZ005109-10

LUZ005118

LUZ006056

LUZ006507-09

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LUZ012732-33

LUZ012863-64

LUZ012865-66

LUZ013053-55

LUZ013093

LUZ013094-95

LUZ013367-87

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LUZ015663

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LUZ023843-35

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PCPC0017629

PCPC0052415

WCD000254-55

After your doctor or health care provider prescribes your ORTHO Diaphragm, 1998. (JANSSEN000056-65)

Agenda: NTP Board of Scientific Counselors Report on Carcinogens (RoC) Subcommittee Meeting

Annie Yessian Report - Echeverria

Berg v. Johnson & Johnson, Final Jury Instructions

Berg v. Johnson & Johnson, Judgment

Berg v. Johnson & Johnson, Verdict Form October 4, 2013

California State Cosmetics Program from the California Dept of Public Health - Occupational health Branch - Chemicals known or suspected to cause cancer or reproductive toxicity (P-31)

California Safe Cosmetics Act 2005

Carl v. J&J; Balderrama v. J&J - Defendants Johnson & Johnson, Johnson & Johnson Consumer Inc., and Imerys Talc America, Inc's joint memorandum of law in support of their motion to exclude plaintiffs' experts' general causation opinions

Cancer Prevention Coalition – November 17, 1994 Citizen's Petition to FDA seeking carcinogenic labelling on all cosmetic talc products

Cancer Prevention Coalition – May 13, 2008 Citizen's Petition to FDA seeking a cancer warning on cosmetic talc products

Cesario, S - Powerpoint "Feminine hygiene product use and the risk of ovarian cancer"

Committee on the State of the Science in Ovarian Cancer Research; Board on Health Care Services; Institute of Medicine; National Academies of Sciences, Engineering, and Medicine. Ovarian Cancers: Evolving Paradigms in Research and Care

Cesario, Sandra. PTT-Feminine hygiene product use and the risk of ovarian cancer (*Unpublished*).

Crowley M. (November 12, 2018) Rule 26 report of Michael M. Crowley, PhD regarding the fragrance chemical constituents in Johnson & Johnson Talcum Powder Products

Daniel Cramer Report - Echeverria

Daniel Cramer Supplemental Report - Echeverria

David Steinberg, expert report

David Steinberg, FRAPS Exhibit 14: Statement of Michael M. Landa, J.D.

David Steinberg, CV

David Steinberg publications list

David Steinberg signed verification Di Saia, P. J. (2015). Letter to Kathleen A. Frazier. Unpublished letter.

Defense Expert Reports from Blaes Case: DeSesso; Hoel; Di Saia; Muscat; Hopkins

Deposition Exhibit of John Hopkins – 28 (November 5, 2018)

Deposition Exhibit of Julie Pier – 47 (September 13, 2018)

Deposition Transcript of Alice Blount, *Ingham v. Johnson & Johnson, et al*. (April 13, 2018)

Deposition Transcript & Exhibits - Julie Pier, MDL No. 2738 (September 12 – 13, 2018)

Deposition transcript, 10/19/2012 - John Hopkins

Deposition Transcript & Exhibits - John Hopkins, MDL No. 2738 (Aug. 16 – 17, 2018, Oct. 17, 2018, Nov. 5, 2018)

Deposition Transcript & Exhibits - Joshua Muscat, MDL No. 2738 (Sept. 25, 2018)

Deposition Transcript & Exhibits - Linda Loretz, MDL No. 2738 (July 17, 2018, Oct. 1 – 2, 2018)

Deposition Transcript & Exhibits - Robert Glenn, MDL No. 2738 (Oct. 18, 2018)

D. L. Longo, R. C. Young. Cosmetic talc and ovarian cancer (1979)

D. L. Longo, R. C. Young. Letter to the Editor: Cosmetic talc and ovarian cancer (1979)

Educational Report of Thomas Dydek

Excerpts from S. Sharma Deposition

Expert Report of Laura M. Plunkett, PhD, DABT – Oct. 5, 2016

Expert Report of Jack Siemiatycki, MSc, PhD – Oct. 4, 2016

Fair warning TalcDoc 5 - Exhibit 113 (JNJNL91_000022019)

Fair warning TalcDoc 15

FDA Authority Over Cosmetics April 6, 2015

FDA Response to Citizen's Petition re: Docket Numbers 94P-0420 and FDA-2008-P-0309-00001/CP

Federal Register – 81 FR 91722 – Banned Devices – Powdered Gloves

Fox v. Johnson & Johnson, Trial Transcript

Godleski, J. J. (2015). Letter to R. Allen Smith, Jr. Unpublished letter.

John J. Godleski, M.D. - Expert Report from Blaes Case

John Godleski Report - Echeverria

John Godleski Supplemental Report - Echeverria

Hopkins, J. (2015). Letter to Gene M. Williams. Unpublished letter.

Johnson's Baby Powder - website, product description

Kemp Hearing Transcript - Douglas Weed

Longo, Rigler, Egeland. MAS Project 14-1852: Below the Waist Application of Johnson & Johnson Baby Powder, September 2017

Longo, Rigler - Analysis of Johnson & Johnson Baby Powder & Valiant Shower to Shower talc products for amphibole (tremolite) asbestos, August 2017

Longo, Rigler - Analysis Report MAS Project #14-1683 Johnson's Baby Powder Sample Set, April 2017

Longo, Rigler - TEM Analysis of historical 1978 Johnson's Baby Powder Sample for amphibole asbestos, February 2018

Longo, Rigler – Expert Report In re: Talcum Power Prod. Liab. Litig., MDL No. 2738 (November 14, 2018).

"Making it up as he goes along: Paolo Boffetta, Italian Epidemiologist, distorts power line health risks" - https://microwavenews.com/news-center/boffetta-post-truth.

Material Safety Data Sheet from Luzenac America, Inc.; Version 45.0, updated 6/18/08 (Group 1)

Material Safety Data Sheet from Luzenac America, Inc. (Group 3)

Material Safety Data Sheet from Luzenac America, Inc.; Version 2.0, updated 2/26/09 (Group CAN)

Material Safety Data Sheet from Luzenac America, Inc. (Group 1)

MBS Invoices – December 2007, April 2012, May 2013, July 2013, December 20013, January 2015, March 2015

MSDS Sheet, Version 2.0

Muscat, J. E. (2015). Report on the Relationship between Hygienic Use of Talc and the Risk of Ovarian Cancer. Unpublished report.

NPR Article Johnson & Johnson Pledges to Purge Controversial Chemicals April 16, 2015

Ness, R. B. (2015). Report on the question of whether genital talc use causes ovarian cancer. Unpublished report.

Ness, R. Expert Report - Jacqueline Fox

Ness, R. Commentary "A plaintiff's witness in the baby powder case"

NTP "The Report on Carcinogens Tenth Edition - Factsheet"

Omiecinski, C. J. (2015). Opinion on the Relationship Between Chronic Perineal/Genital Exposures to Cosmetic Talc and Ovarian Cancers: Mechanistic Aspects and Biological Plausibility Unpublished report.

Osann, K. (2016). Report on Perineal Talc Exposure and Risk of Ovarian Cancer. Unpublished report.

Personal Care Products Council Letter – July 21, 2009 to FDA re: Comments to FDA Citizen's Petition to the Commissioner of the Food and Drug Administration Seeking a Cancer Warning on Talc Products

Photographs of Johnson's Baby Powder

Photographs of Shower to Shower

Riham Sheble, Shabina Khatri. DOHA News - Johnson's baby powder of Qatar shelvees after US cancer lawsuit verdict

Roe. Controversy: Cosmetic tlac and ovarian cancer (1979)

Rosenthal, G. J. (2015). Opinion on Relationships Between the Toxicology of Cosmetic Talc and the Pathogenesis of Ovarian Cancer. Unpublished report.

Rosenthal, G - Expert Report from Blaes Case: "Opinion on Relationships Between the Toxicology of Cosmetic Talc and the Pathogenesis of Ovarian Cancer"

Rothman, Pastides, Samet. (2000) Interpretation of epidemiologic studies on talc and ovarian cancer

Slemp v. Johnson & Johnson, et al., Trial Transcript

Summary Minutes of the NTP Board of Scientific Counselors Report on Carcinogens (RoC) Subcommittee Meeting

Talc Removed from 12th RoC- The Rose Sheet October 24, 2005

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Weed, Douglas. A Report Regarding General Causation and an Evaluation of the Reliability and Validity of the Plaintiffs' Experts' Reports Designated for the Plaintiff, Lori Oules (Feb. 1, 2017)

17. Curriculum Vitae – Jack Siemiatycki

CURRICULUM VITAE

Jack Siemiatycki

TABLE OF CONTENTS

STATISTICAL SUMMARY OF SELECTED ACCOMPLISHMENTS

Publications in peer-reviewed journals	245
Book chapters, IARC Monographs	20
Other publications, reports	42
Book (authored)	1
Invited presentations	173
Conference presentations, posters, abstracts : offered and accepted	181
Grants received as P.I. (number)	36
Grants received as P.I. ($)	$15.4M
Grants received as co-investigator (number)	59
Grants received as co-investigator ($)	$27.9M
H-factor (google scholar)	64
Instances of participation on expert panels, committees, boards of directors, at invitation of governments or public health agencies or research agencies or universities	126
Grant review panels or referee for external institution or journal editorial boards	65
Honours	several

GENERAL INFORMATION

Work address
Université de Montréal
Research Center of CHUM
850 rue St Denis, Montréal, QC, Canada H2W 1V1

Tel: (514) 890-8166
Fax: (514) 412-7106
E-mail: j.siemiatycki@uMontréal.ca

EDUCATION

1967 B.Sc. (mathematics); McGill University
1970 M.Sc. (mathematical statistics); McGill University
1976 Ph.D. (epidemiology and medical statistics); McGill University
1977 Post-doctoral (cancer epidemiology); International Agency for Research on Cancer, Lyon

CURRENT ACADEMIC APPOINTMENTS

Professor, Department of Social and Preventive Medicine, Université de Montréal (since 2001)
Cancer Research Society-Guzzo Research Chair in Environment and Cancer, Université de Montréal (since November 2007)
Adjunct Professor, Department of Epidemiology, Biostatistics and Occupational Health, McGill University. (since 1979)
Fellow, Canadian Academy of Health Sciences (since 2008)

PREVIOUS ACADEMIC APPOINTMENTS AND WORK EXPERIENCE

1967-71	Research Fellow; Department of Epidemiology and Health, McGill University.
1970-72	Research Director; Pointe St. Charles Community Clinic, Montréal.
1978	Consultant; International Agency for Research on Cancer, Lyon.
1978-2001	Assistant, then Associate (1979), then full Professor (1983): Epidemiology Research Center, Institut Armand-Frappier, Laval, Québec.
1982-1986	Associate member, McGill Cancer Center, McGill University.
1996-1997	Visiting Scientist. International Agency for Research on Cancer, Lyon.
2001-2015	Canada Research Chair (Tier 1), Université de Montréal (resigned 2011).
2003-2009	Affiliate Scientist. McLaughlin Centre for Pop'n Health Risk Assessment, Univ of Ottawa.

SIGNIFICANT INTERNAL ADMINISTRATIVE APPOINTMENTS

1982-86	Director, Équipe associée de l'Institut de Recherche en Santé et Sécurité du Travail sur les cancers professionnels (affiliated research team of the Quebec Institute for Occupational Health and Safety on Occupational Cancer).
1988-91	Director, Epidemiology Research Center, Institut Armand-Frappier.
1990-98	Director, Équipe prioritaire de recherche en épidémiologie environnementale du FRSQ. (Priority research team in environmental epidemiology)
1998-2001	Member, Governing Council (Conseil d'administration). Institut national de la recherche scientifique, Université du Québec.
2000-2007	Coordinator. Program of Research in Environmental Epidemiology of Cancer (PREECAN), a national program funded by the National Cancer Institute of Canada.
2002-2005	Associate Director for Population Health Sciences, Research Center of the University of Montréal Hospital Center.

2006-2007 Director, Epidemiology program, PhD public health, Université de Montréal.
2006-2014 Director, Axe risques à la santé (Health Risks Division). Centre de recherche du Centre hospitalier de l'Université de Montréal.

SIGNIFICANT INSTITUTIONAL COMMITTEES

1979-80 Member of faculty committee to negotiate a collective agreement with the Institut Armand-Frappier administration.
1982-92 Member, Research Council. Institut Armand-Frappier.
1998-2001 Member, Institutional advisory council. Institut Armand-Frappier. Institut national de la recherche scientifique
2002-2006 Comité de direction. Centre de recherche du CHUM
2002-2017 Member, Various committees of the Dept Med Soc et Preventive, including Promotions, and Recruitment.
2006-2009 Member, Various committees established to set up a new School of Public Health at l'Université de Montréal
2006-2014 Comité Scientifique de la Recherche du CHUM.

CURRENT MAJOR EXTERNAL BOARDS, SCIENTIFIC COMMITTEES (INVITED)

1. Chair of Scientific Advisory Committee of CONSTANCES, a large prospective cohort established in France, under aegis of INSERM, Ministère de la Santé, and other agencies. Since 2011.
2. Member of Comité national d'épidémiologie en cancérologie. Ministère de la Santé et des Services sociaux, Quebec. Since 2014.
3. Member, Advisory committee to Directors of Cartagene, a Quebec population cohort.

PAST MAJOR EXTERNAL BOARDS, SCIENTIFIC COMMITTEES, CONSULTATIONS (INVITED)

1. Expert consultative committee to Commission de la santé et sécurité du travail du Québec on the epidemiologic function of the CSST. 1979-80.
2. President of Organizing Committee of Annual Congress of Quebec Public Health Association, Montréal. 1982.
3. Consultative committee of International Agency for Research on Cancer on feasibility of SEARCH programme. 1982.
4. Canadian representative. International Joint Commission (U.S. and Canada) Committee on the Assessment of Human Health Effects of Great Lakes Water Quality. 1982-89.
5. Task Force on Chemicals in the Environment and Human Reproduction Effects in New Brunswick. 1983-85.
6. Chairman and organizer of international workshop sponsored by International Agency for Research on Cancer, Lyon, on use of job exposure information in cancer case-control studies. 1984.
7. Quebec Government Consultative Committee on Alachlor. 1985-86.
8. Chairman and organizer of the International Joint Commission Workshop on the Role of Epidemiology in Assessing the Effects of Great Lakes Water Quality on Human Health, Scarborough, March 1988. 1986-88.
9. Priority Substances Advisory Panel. Panel established under terms of Canadian Environmental Protection Act by Health and Welfare Canada. 1988.
10. Working Group on Electromagnetic Fields under auspices of Health Effects Institute. 1991.
11. Consultative Committee on Environment-related Cancer Surveillance, LCDC, Health and Welfare Canada. 1993-1996.
12. Consultative Committee on an Investigation of Lung Cancer and Environmental Tobacco Smoke, Environmental Health Directorate, Health Canada. 1994-1995.
13. Working Group on Evaluation of Carcinogenicity of Carbon Black, Printing Trades and Various Substances. Monograph Programme. International Agency for Res. on Cancer, Lyon, 1995.

14. Working Group on Human Cancer Risks associated with Chrysotile Asbestos. World Health Organization (IPCS) Geneva, June 1995.
15. Secretariat on Evaluation of Chemopreventive Effect of Aspirin and Other NSAIDS for Cancer. International Agency for Res. on Cancer, Lyon, Apr. 1997.
16. Chair. Symposium on Health Risks of Water Disinfection By-products. Convened by Health Canada. Ottawa. May 1997.
17. Working Group. Meeting on Species-specificity in response to carcinogens. Monograph Programme. International Agency for Res. on Cancer, Lyon, Nov. 1997.
18. Board of Directors. Canadian Society for Epidemiology and Biostatistics. 1997-1999.
19. Working Group. Evaluation of Carcinogenicity of Various Industrial Substances. Monograph Programme. International Agency for Res. on Cancer, Lyon, Feb. 1998.
20. Canadian Coalition on Cancer Surveillance. 1997-2002.
21. External site review panel. U.S. National Cancer Institute Epidemiology Branch. June 1999.
22. Organizing Committee for Medical Research Council Workshop on Privacy of Health Data. 1999-2000.
23. Organizing Committee, EPI2001. Joint North American Congress of Canadian Society for Epidemiology and Biostatistics, Society for Epidemiologic Research, American Public Health Association (Epid) and American College of Epidemiology, Toronto, 14 – 16 June 2001. 1999-2001.
24. Coordinator of national initiative of the public health community to provide guidance on the structures and functioning of the new Canadian Institutes of Health Research. 1999-2000.
25. Organizing Committee. World Congress of the International Epidemiological Association, Montréal, 18-22 August 2002. 2001-2002.
26. President. Canadian Society for Epidemiology and Biostatistics. 2001-2003. Member of Board. 1997-1999.
27. Working Group. Evaluation of Carcinogenicity of Various Substances. Monograph Programme. International Agency for Res. on Cancer, Lyon, 2003.
28. Jury of Consensus Conference on risks and benefits of vaccination for hepatitis B. For Minister of Health of France. Organized by INSERM and ANAES. Paris 2003.
29. Public Advisory Panel. Vinyl Council of Canada. 1998-2004.
30. Advisory Panel. U.S. National Cancer Institute Brain Tumor Study. 1998-2003.
31. Scientific Advisory Committee. Boeing/UAW Workers' Health Studies. 1999-2005.
32. Institute Advisory Board. Canadian Institutes for Health Research – Institute of Circulatory and Respiratory Health. 2001-2005.
33. National Occupational Research Agenda (NORA). Joint consultative committee for US National Cancer Institute and US National Institute for Occupational Safety and Health. 2002-2005.
34. Canadian Cancer Surveillance Alliance. Consultative committee of Health Canada, Canadian Cancer Society, Provincial Cancer Registries, Statistics Canada. 2002-2003.
35. Co-president. Organizing Committee of Joint SER-CSEB Congress, Toronto 27-30 June 2005. (2004-2005).
36. Chair. Monograph Program Meeting. International Agency for Research on Cancer (WHO), France. February 2006.
37. Advisory Committee on Research Ethics and Databanks. Quebec Health Research Council (FRSQ). 2003-2011.
38. Board of Directors. American College of Epidemiology. 2003-2006.
39. Board of Directors. National Cancer Institute of Canada. 2003-2007.
40. Member Scientific Council International Agency for Research on Cancer (WHO). Lyon, France 2005-2009.
41. Elected Chair. Scientific Council International Agency for Research on Cancer (WHO). Lyon, France 2008-2009.
42. Scientific Advisory Council Canadian Partnership Against Cancer 2007-2009.
43. Advisory Committee. Occupational Cancer Research Centre of Ontario. Since 2009.
44. Working Group on Cancer Prevention, CPAC, 2007-2010.

45. Subgroup Chair and Working Group Member. Evaluation of Carcinogenicity of Non-Ionizing Radiation, Radiofrequency Electromagnetic Fields. Monograph Programme. International Agency for Research on Cancer, Lyon May 2011.
46. Member of Scientific Advisory Board of Bordeaux cancer research center SIRIC-BRIO, Bordeaux France. Since 2013.
47. Member of external review panel. Helmholtz Center Munich Research Institute. Germany. July 2011.
48. Conseil Scientifique de l'Institut de Recherche en Santé Publique (IReSP). Under aegis of INSERM and Ministère de la Santé, France. 2004-2009.
49. Adviser and expert witness for legal team conducting a major class action lawsuit against the Canadian tobacco industry. 2007-2014.

OTHER SIGNIFICANT EXTERNAL CONSULTATIONS (INVITED)

1. Consultation with Quebec Ministry of Justice regarding compensation for homeowners who were advised to use formaldehyde-base home insulation - 1983.
2. Invited participant. Workshop convened by the Science Council of Canada on the future of Epidemiology in Canada, Ottawa - 1985.
3. Consultation with Government of Alberta regarding the evaluation of a report alleging significant health impact in the environment of a sour-gas plant - 1985.
4. Consultation with Quebec Ministry of Environment regarding health effects of residency near an abandoned toxic waste site in LaSalle, Quebec - 1987.
5. Invited participant. Workshop convened by Canadian Public Health Association, Environment Canada and Health and Welfare Canada on Environmental Impact Assessment, Ottawa - 1987.
6. Invited participant. Annual workshops convened by Health Protection Branch of Health and Welfare Canada to discuss the role of Canada in the SEARCH programme of the International Agency for Research on Cancer, Ottawa - 1987-1989.
7. Consultation with Quebec Cree Band Council regarding a research proposal to study developmental effects of consuming fish with high mercury levels - 1989.
8. Invited participant. Workshop convened by Ontario Industrial Disease Standards Panel on the use of epidemiologic data in workers' compensation, Toronto - December 1989.
9. Invited participant. Workshop convened by National Academy of Sciences (U.S.) on Carcinogenicity of Complex Mixtures, Tucson, Arizona - Jan 1990.
10. Invited participant. Workshop convened by Laboratory Centers for Disease Control, Health and Welfare Canada on Multiple Chemical Sensitivities, Ottawa - May 1990
11. Member of expert advisory panel to the pan-Canadian case-control study of electromagnetic fields and childhood leukemia. Sponsored by Canadian Electrical Assoc, EPRI (U.S.A.), Health and Welfare Canada. 1990-1996.
12. Organizer of Workshop to Plan a Pan-North American Case-control Study of Lung Cancer. Sponsored by Health and Welfare Canada. Toronto. March 1991.
13. Invited participant. Workshop convened by Environmental Health Directorate of Health and Welfare Canada, on Environmental Epidemiology in Canada. Ottawa. March 1992
14. Invited participant. Workshop convened by Harvard Center for Risk Analysis on implementing a new type of risk assessment. Maryland. April 1992.
15. Member of Technical Advisory Panel for epidemiology studies of foundry workers - CIIT. Research Triangle Park, N.C. Feb. 1993
16. Consultant to Health Effects Institute - Asbestos Research, on Options for Characterizing Worker Activities in Buildings, Boston. Feb. 1993.
17. Advisory panel to Laboratory Centers for Disease Control, Health and Welfare Canada, on Environmental Epidemiology under the Green Plan. March 1993.
18. Member of External Advisory Committee. Champlain Adirondack Biosphere Environmental Health Sciences Center, University of Vermont. 1993.
19. Consultant to Michigan Cancer Foundation on a variety of epidemiologic studies. 1993-1996.

20. Invited to address President Clinton's Panel on Cancer regarding priorities in cancer research. Bethesda, MD. April 1994.
21. Invited participant. Science and Technology Review Consultation. Government of Canada. Montréal. September 1994.
22. Invited participant. Strategic planning workshop to reduce Environmental Tobacco Smoking exposure. Laboratory Centre for Disease Control. Health Canada. Oct 1995.
23. Invited participant. Meeting to establish new priorities for funding. National Health Research and Development Programme of Canada. Montréal. Feb 1996.
24. Chair Scientific Advisory Committee for the Dalhousie University study of health effects of environmental and occupational pollution in the area of the Sydney, Nova Scotia steel industry. 1996.
25. Member of two Ministerial missions of the Quebec and Canadian governments to France to discuss with French experts the risks associated with low level exposure to chrysotile asbestos. Paris. Oct 1996.
26. Chair. Meeting of collaborators of European network of studies on lung cancer and smoking.
27. International Agency for Res. on Cancer, Lyon. June 1997.
28. Member of Canadian scientific delegation to United Kingdom to discuss with British experts the risk associated with low level exposure to chrysotile asbestos. London, Sept. 1997.
29. Symposium chair. Workshop to discuss methods of predicting numbers of cases of mesothelioma to be expected in various countries. Paris. Dec. 1997.
30. Invited participant and subgroup reporter. Peer Review on Hazard Assessment and Dose-Response Characterization for the Carcinogenicity of Formaldehyde by the Route of Inhalation. Health Canada and U.S. EPA. Ottawa. March 1998.
31. Co-chair. Workshop to explore the feasibility of an international collaborative study on use of cellular phones and risk of cancer. International Agency for Research on Cancer. Lyon. Feb 1999.
32. Panellist. Consensus Meeting for a Proposed Integrated National Health Surveillance Network. Health Canada. 1999.
33. Invited participant. Medical Research Council Summit Meeting on the new Canadian Institutes of Health Research. Toronto. June, 1999.
34. Invited participant. Planning group for an Institute of Population Health Research in CIHR. Jul-Dec 1999.
35. Invited speaker. Workshop for a Canadian Institute for Genetics Research. May 2000.
36. Invited participant. Workshop to explore the use of prospective cohorts to investigate gene-environment interactions in cancer etiology. National Cancer Institute. Rockville, MD. May 2000.
37. Invited participant. Founding meeting of Canadian Association for Workplace Safety and Health. Montréal. Jan 2001.
38. Invited participant. Workshop to advise Canadian Foundation for Innovation on its role in supporting population health research in Canada. Toronto, Feb 2001.
39. Invited participant. Consultative committee to advise Cancer Care Ontario on priorities in environmental cancer. April 2001.
40. Invited participant. Workshop on national priorities in cancer research. Institute for Cancer Research. CIHR. Toronto. May 2001.
41. Invited participant. Delphi process to advise Canadian Institutes of Health Research on priorities in cancer research. October-December 2001.
42. Invited participant. Delphi process to advise Cancer Care Ontario on priorities in cancer prevention. November-April 2002.
43. Session Chair. NIOSH workshop "Applying New Biotechnologies to the Study of Occupational Cancer", Washington, D.C. May 2002.
44. Member of Advisory Panel. U.S. National Cancer Inst. Study of a Cohort of Chinese Workers Exposed to Benzene. 2002- .
45. Invited participant. Delphi process to advise Cancer Care Ontario on priorities in cancer prevention. November-April 2002.
46. Session Chair. NIOSH workshop "Applying New Biotechnologies to the Study of Occupational Cancer", Washington, D.C. May 2002.

47. Organizer and Session Chair. International Epidemiological Association Meeting. Occupation and Health. Montréal. August, 2002.
48. Co-Organizer and Session Chair. Epidemiological Association Meeting. Asbestos and mesothelioma. Montréal. August, 2002.
49. Session Chair. Epidemiological Association Meeting. Environment and Health. Montréal Aug, 2002.
50. Invited participant. CIHR National Forum to devise a National Research Programme for Environmental Health. Ottawa. Sept 2002.
51. Invited participant. CIHR national forum on privacy of health data. Ottawa, November, 2002.
52. Member. Environmental and Occupational Carcinogens Advisory Group. Canadian Cancer Society. 2002 - 2004.
53. Participant. Meeting to discuss the establishment of a prospective childhood cohort in Canada. CIHR-IPPH. March 2004.
54. Member of working group on national cohort project. National Cancer Research Initiative. January-June 2004.
55. Member of advisory group on development of IDEES, Université de Montréal. January-June 2004.
56. Member, ad-hoc group to explore the feasibility of a Canadian cohort on cancer and chronic disease. 2004-2008.
57. Invited participant. Workshop to discuss the enhancement of population health research in Canada. CIHR-IPPH. June 2004.
58. Invited participant. Workshop on occupational cancer surveillance. Occupational Cancer Research & Surveillance Project (Cancer Care Ontario and the Ontario Workplace Safety & Insurance Board). February 2005.
59. Invited participant. Workshop on long-term large-scale cohorts. CIHR, December 2005.
60. Member. Advisory Scientific Committee. IBM – University of Alabama project on health of IBM manufacturing plant workers. 2006 - 2008.
61. Advisor and meeting participant. Ontario Workplace Safety and Insurance Board. Recommendations on how to develop occupational cancer research in Ontario. Toronto, 2005.
62. Invited participant. Workshop to estimate the burden of occupational cancer in the United Kingdom. UK Health and Safety Executive. Manchester. June 2006.
63. Advisory Committee to British Energy Networks Association. Workshop on the Future Needs of Electro-magnetic Fields Occupational Studies in the Electric Utility Industry. Edinburgh. September 2006.
64. Advisory Committee. IARC Monograph Programme Planning of Special Volume 100. Lyon. September 2006.
65. Grant Review Panel. IVRSP. Paris. September 2006.
66. Advisory Committee to CCRA and ICR (CIHR) on the nature of a national cohort platform. Toronto, September 2006.
67. Invited participant. Comité d'éthique de la recherche de la faculté de médecine (CERFM) : Discussion d'un projet soumis pour la création d'une banque de données et de matériaux biologiques (Research Ethics Committee of the Faculty of Medicine: Review of a submitted project to create a bank of data and biologic samples). Université de Montréal. March 2007.
68. Invited participant. Workshop to Design and Implement the Ontario Cohort Consortium Research Platform. Toronto. June 2007.
69. Invited participant. Canadian Cancer Research Agencies. Strategic Planning Consultation in Montréal. May 2009.
70. Invited participant. IARC-NORA workshop to identify gaps of knowledge on occupational carcinogens, Lyon. June 2009.
71. Consultant. State of the science workshop: evaluation of epidemiological data consistency for application in regulatory risk assessment. US EPA and Johns Hopkins School of Public Health. Baltimore. September 2010.
72. Consultant. World Health Organisation. Re-evaluation of Risk Assessments related to DDT exposure. Geneva. November 2010.

73. Invited participant. WHO workshop to develop international guidelines for control of environmental carcinogens. Asturias. March 2011.
74. Session Chair. Discovering occupational carcinogens. Congress of Epidemiology. Montréal June 2011.
75. Invited co-organiser. Symposium of Environment and Cancer. Canadian Cancer Research Conference. Toronto. November 2011.
76. Invited organiser and Chair. Symposium on Cellphones and Cancer. American Association for Cancer Research. Chicago, April 2012.
77. Member Scientific Program Committee for the 2013 Canadian Cancer Research Conference, Toronto. November 2013.
78. Member of Advisory Committee to National Cancer Institute (U.S.) study on carcinogenicity of diesel emissions. 2017.

HONOURS

1. Biographee in various Who's Who in America versions. Since 1982
2. Perron-Desrosiers Prize. Granted by the Governing Council of the Institut Armand-Frappier. 1985.
3. Invited to give the annual Elizabeth Stern Memorial Lecture in U.C.L.A. School of Public Health. 1985.
4. National Health Scholar. National Health Research and Development Programme of Canada. 1988-1998.
5. Visiting Scientist Award. International Agency for Research on Cancer, Lyon. 1996-97.
6. Prix d'excellence. Institut national de la recherche scientifique. Université du Québec. 1999.
7. Distinguished Scientist Award. Medical Research Council, Canada. 1999-2004.
8. Canada Research Chair in Environmental Epidemiology and Population Health. 2001-2015.
9. Distinguished Scientist Lecturer. US National Cancer Institute. Division of Cancer Epidemiology and Genetics. 2006.
10. Cancer Research Society–Guzzo Chair in Environment and Cancer. Since 2007.
11. Fellow Canadian Academy of Health Sciences. Since 2008.
12. Geoffrey R Howe Distinguished Contributions Award, Canadian Society for Epidemiology & Biostatistics. 2011.
13. Ranked top Canadian public health researcher in terms of research productivity by Jarvey et al. 2012.

GRANT REVIEW, JOURNAL REVIEW AND PERSONNEL REVIEW

<u>Associate Editor</u>

American Journal of Epidemiology (1989-1998)
International Journal of Environmental Health (1991-)

<u>Contributing Editor</u>

Journal of Public Health Policy (1982-87)
American Journal of Industrial Medicine (1996-)
The Open Epidemiology Journal (2007-)

<u>Chairman of grant review panels</u>

National Health Research and Development Programme. Canada. (1990-94)
National Cancer Institute of Canada (1994-1995)

<u>Member of grant review panels</u>

40 times

<u>External referee for tenure or promotion of personnel in other institutions</u>

15 times

THESES

1. Siemiatycki J. “Space-time clustering: finding the distribution of a correlation-type statistic”. M.Sc. thesis, McGill University, 1971.
2. Siemiatycki J. “Evaluation of strategies for household health surveys”. Ph.D. thesis, McGill University, 1976.

ARTICLES PUBLISHED PEER REVIEW

1. Thurlbeck WM, Horowitz I, Siemiatycki J, Dunnill MS, Maisel JC, Pratt P, et al. Intra- and inter-observer variations in the assessment of emphysema. Archives of Environmental Health. 1969;18:646-59.
2. Becklake MR, Fournier-Massey G, McDonald JC, Siemiatycki J, Rossiter CE. Lung function in relation to chest radiographic changes in Quebec asbestos workers. Bulletin de Physio-Pathologie Respiratoire. 1970;6:637-59.
3. McDonald JC, McDonald AD, Gibbs GW, Siemiatycki J, Rossiter CE. Mortality in the chrysotile asbestos mines and mills of Quebec. Archives of Environmental Health. 1971;22:677-86.
4. Siemiatycki J, McDonald AD. Neural tube defects in Quebec: a search for evidence of `clustering' in time and place. British Journal of Preventive and Social Medicine. 1972;26:10-4.
5. Siemiatycki J. Mantel's space-time clustering statistic: computing higher moments and a comparison of various data transforms. Journal of Statistical Computation & Simulation. 1978;7:13-31.
6. Siemiatycki J. A comparison of mail, telephone, and home interview strategies for household health surveys. American Journal of Public Health. 1979;69(3):238-45.
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9. Siemiatycki J, Richardson L, Pless IB. Equality in medical care under national health insurance in Montréal. New England Journal of Medicine. 1980;303:10-5.
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69. Siemiatycki J. Occupational cancer. Seminar series of Laboratory Centre for Disease Control, Health and Welfare Canada, Ottawa, March 1991.
70. Siemiatycki J. A decade of searching for occupational carcinogens: methods and results of a case-control study. Seminar series of the Division of Clinical Epidemiology, Montréal General Hospital, Montréal, March 1991.
71. Siemiatycki J. Detecting occupational carcinogens using epidemiologic methods: results and their interpretation. McGill University, Department of Epidemiology and Biostatistics, Summer Lecture Series, Montréal, June 1991.
72. Siemiatycki J. Overview of results of an occupational cancer monitoring study. School of Public Health, University of California at Berkeley, Berkeley, October 1991.

73. Siemiatycki J. Discussant of paper on Mortality of oil refinery and distribution workers. International Symposium on the Health Effects of Gasoline, Miami, November 1991.
74. Begin, D, Gerin M, De Guire L, Siemiatycki J, Adib G, Fournier C. Étude sur la validité des matrices emploi-exposition multisectorielles en hygiène industrielle. Scientific Committee on Computing in Occupational and Environmental Health, III International Workshop, Paris, November 1991.
75. Siemiatycki J. Cancer et travail : connaissances actuelles, approches antérieures et nouvelles. Colloque de l'Association des médecins du travail du Québec, Montréal. June 1992.
76. Siemiatycki J. Risques de cancers reliés aux expositions chimiques en milieu de travail: résultats d'une étude épidémiologique à Montréal. IRSST, Montréal, November 1992.
77. Siemiatycki J. Carcinogens in the occupational environment. Invited seminar in School of Public Health, University of North Carolina, Chapel Hill, North Carolina. December 1992.
78. Siemiatycki J. Discussant of invited seminar on risk assessment. School of Occupation Health, McGill University, March 1993.
79. Siemiatycki J. Are the effects of smoking on lung and bladder cancer confounded by occupational carcinogens? Invited seminar given at the Michigan Cancer Foundation, Detroit and at the University of Michigan, Ann Arbor, May 1993.
80. Siemiatycki J. Are the apparent effects of cigarette smoking on lung and bladder cancers due to uncontrolled confounding by occupational exposures? McGill University, Department of Epidemiology and Biostatistics, Montréal, December, 1993.
81. Siemiatycki J. Occupational causes of cancer. President's Cancer Panel Meeting on Avoidable Causes of Cancer, Bethesda, April 1994.
82. Siemiatycki J. Retrospective exposure assessment in community-based studies. Conference on Retrospective assessment of occupational exposures in epidemiology, IARC, Lyon, April 1994.
83. Siemiatycki J. Are the apparent effects of cigarette smoking on lung and bladder cancers due to uncontrolled confounding by occupational exposures? Department of Human Oncology, University of Torino, Torino, Italy, April 1994.
84. Siemiatycki J. Risque de cancer dû au tabagisme. Département de médecine sociale et préventive, Université Laval, Québec, May 1994.
85. Siemiatycki J. Registry studies of bladder cancer. NCI Workshop on Occupational Exposures and Urogenital Cancers, Rockville, May 1994.
86. Siemiatycki J. Facteurs de risques environnementaux pour le cancer: une perspective épidémiologique. Atelier sur la recherche en cancer, Université du Québec à Rimouski, April 1995.
87. Camus M, Siemiatycki J. Non-occupational exposure to asbestos: how to assess dose and risk. McGill University, Department of Epidemiology and Biostatistics. Montréal, May 1995.
88. Siemiatycki J. Occupational carcinogens in Montréal. Seminar - International Agency for Research on Cancer, Lyon, France, June 1995.
89. Siemiatycki J. Occupational causes of cancer: findings from a large scale case-control study of occupational cancer. Department of Medical Informatics, Biometry and Epidemiology, University of Essen, Essen, Germany, July 1995.
90. Siemiatycki J. Assessing occupational exposures in community based epidemiological studies. Bremen Institute for Preventive and Social Medicine, Bremen, Germany, July 1995.
91. Case, B, Camus M, Richardson L, Siemiatycki J. Ascertainment of mesothelioma among Québec women from 1970 to 1990. Special Symposium on Mesothelioma, IRSST, Montréal, August 1995.
92. Siemiatycki, J. Une nouvelle approche épidémiologique pour le dépistage de cancérogènes en milieu de travail. Club de recherches cliniques du Québec, Bromont, Quebec, September 1995.
93. Siemiatycki J. Occupational causes of cancer: findings from a large scale case-control study of occupational cancer. Special seminar. School of Public Health, Univ. of Michigan, Ann Arbor, Michigan, June 1996.
94. Siemiatycki J. An empirical evaluation of the magnitude of confounding bias. Statistical Society of Canada, Waterloo, June 1996.

95. Siemiatycki J. Occupational exposures and cancer risk: recent results and methodological insights from a population-based case-control study in Montréal. Department of Epidemiology & Biostatistics, McGill University. October, 1996.
96. Siemiatycki J. Utilités et limites des études épidémiologiques dans l'évaluation des risques environnementaux. ACFAS, City of Québec, Quebec, May 1998.
97. Siemiatycki J. International collaboration in cancer epidemiology. Society for Epidemiology Research, Chicago, June 1998.
98. Siemiatycki J. Accuracy of the EPA risk assessment model for predicting the risk of lung cancer at environmental levels of asbestos exposure. National Cancer Institute, Rockville, Maryland, March 1999.
99. Siemiatycki J. Risk of lung cancer at environmental levels of asbestos exposure. University of Toronto, Toronto, September 1999.
100. Siemiatycki J. Estimating risks due to low level exposures. Society for Epidemiology Research, Seattle, June 2000.
101. Siemiatycki J. Debater on the proposition that research is a top priority in occupational cancer prevention. Preventive Oncology Seminar, Cancer Care Ontario, Toronto, April 2001.
102. Rachet B, Siemiatycki J, Abrahamowicz M, Leffondre K. Various aspects of smoking behavior on lung cancer risk: a flexible modeling approach. National Cancer Institute, Bethesda, May 2001.
103. Siemiatycki J. Challenges to epidemiology and challenges to Canadian epidemiologists. National Student Conference of Epidemiology, Toronto, June, 2001.
104. Siemiatycki J. President's address. Congress of Epidemiology, Toronto, June, 2001.
105. Siemiatycki J. Découvrir les cancérigènes dans l'environnement: bilan des activités de recherche passées et perspectives d'avenir. Département de médecine sociale et préventive, Université de Montréal, October 2001.
106. Siemiatycki J. Risque de cancer chez les femmes résidantes des villes des mines d'amiante québécoises: Évaluation du risque attribuable à des « faibles » niveaux d'expositions et validation de la méthode d'évaluation de risque (« risk assessment ») du E.P.A. Département de santé environnementale, Université de Montréal, October 2001.
107. Rachet B, Siemiatycki J, Leffondre K, Abrahamowicz M. Relations dose-réponse entre la fumée de cigarette et le cancer pulmonaire à partir d'une étude cas-témoins à Montréal : Estimations utilisant une modélisation flexible. Congrès INRS-Institut Armand-Frappier, Sainte-Adèle, Quebec, November 2001.
108. Siemiatycki J, Camus M, Case B, Desy M, Parent, M.-É. Risque de cancer chez les résidantes des villes de l'amiante au Québec: Évaluation du risque attribuable à des « faibles » niveaux d'expositions et validation de la méthode d'évaluation de risque de l'E.P.A. Symposium sur l'amiante of Institut national de santé publique du Québec, Montréal, December 2001.
109. Laplante O, Parent M.-É, Siemiatycki J. Risque de mésothéliome et de cancer du poumon associé à l'exposition professionnelle aux fibres d'amiante, Montréal 1979-85. Symposium de l'Institut national de santé publique du Québec, Montréal, December 2001.
110. Siemiatycki J. Occupational causes of cancer: overview of the contribution of a study in Montréal, Research Day at Dept of Epidemiology and Community Medicine, University of Ottawa, April 2002.
111. Siemiatycki J. Biostatistical problems in epidemiologic case-control studies. Statistical Society of Canada, Hamilton, Ontario, May 2002.
112. Leffondre K, Abrahamowicz M, Siemiatycki J. Definition of risk sets for Cox's analysis of case-control data with time-varying exposures: A simulation study. Intended Society for Clinical Biostatistics (ISCB), Dijon, France, September 2002.
113. Siemiatycki J. Occupational causes of cancer. CCERN and Health Canada Research Workshop, Montebello, Quebec. October 2002.
114. Siemiatycki J. Occupational causes of cancer. Departmental seminar, McGill University, Montréal. November 2002.
115. Siemiatycki J. Facteurs environnementaux dans l'étiologie du cancer. Retraite annuelle du centre de recherche du CHUM, St-Sauveur, Quebec. November 2002.
116. Siemiatycki J. Environmental and occupational causes of cancer. Seminar. Cancer Care Ontario, Toronto, February 2003.

117. Siemiatycki J. The state of epidemiology in Canada. Plenary address. CSEB Student Congress, Halifax, Nova Scotia, June 2003.
118. Siemiatycki J. Occupational cancer epidemiology: the evolving big picture. Distinguished Scientist Lecture, Division of Cancer Epidemiology and Genetics, National Cancer Institute, Rockville MD, October 2003.
119. Siemiatycki J. Challenges in cancer epidemiology. Meeting of the Institute Advisory Board of Institute for Cancer Research, CIHR, Montréal, June 2004.
120. Siemiatycki J. Keynote address. Occupation and cancer. International Association of Cancer Registries, Beijing, September 2004.
121. Siemiatycki J. Which cancers are most important, what are the associated occupational situations and which confounders are involved? Burden of Cancer Epidemiologic Workshops, Health and Safety Executive. Manchester, UK, November 2004.
122. Siemiatycki J. Occupational causes of cancer. New Strategies for Recognizing and Preventing Occupational Disease, Canadian Center for Occupational Health and Safety, Toronto, March 2005.
123. Siemiatycki J. Occupational causes of cancer. The Respiratory Epidemiology & Clinical Research Unit, Montréal Chest Institute, Montréal, March 2005.
124. Siemiatycki J. Environnement et cancer : quels sont les risques? Les Belles Soirées public lecture series, Université de Montréal, Montréal, April 2005.
125. Siemiatycki J. An overview of environmental, occupational & lifestyle causes of lung cancer. Cancer Axis, McGill University Hospital Centre Research Institute, Montréal, June 2005.
126. Siemiatycki J. Les règles des comités d'éthique vont amputer notre capacité de prévenir des maladies et sauver des vies. Réunion de FRSQ sur les banques de données et des matières biologiques, Montréal, June 2005.
127. Siemiatycki J. Introductory comments. Joint Meeting of the Canadian Society for Epidemiology and Biostatistics and the Society for Epidemiologic Research, Toronto, June 2005.
128. Siemiatycki J. The burden of occupational cancer on workers and on society. Joint Meeting of the Canadian Society for Epidemiology and Biostatistics and the Society for Epidemiologic Research, Toronto, June 2005.
129. Siemiatycki J. Revue des expositions professionnelles associées au cancer (Review of occupational exposures associated with cancer): pre-conference training session. Environnement et santé. Congrès international de l'Association des Épidémiologistes de Langue Française (ADELF), City of Québec, Quebec, September 2005.
130. Siemiatycki J. Opening session. Environnement et santé. Congrès international de l'Association des Épidémiologistes de Langue Française (ADELF), City of Québec, Quebec, September 2005.
131. Siemiatycki J. Impact de l'environnement et du milieu de travail sur le cancer : connaissances récentes. 9es Journées annuelles de santé publique (JASP), City of Québec, Quebec, November 2005.
132. Siemiatycki J. La recherche épidémiologique sur le cancer. Canadian Cancer Society - 2005 Annual Conference, City of Québec, Quebec, November 2005.
133. Siemiatycki J. Occupational EMF exposure and risk of cancer – methodological considerations. Workshop on the Future Needs of Electro-magnetic Fields Occupational Studies in the Electric Utility Industry, Edinburgh, September 2006.
134. Siemiatycki J. What is known about the modifiable causes of cancer and why we will not learn much more: Reflections on the decline of epidemiology as a tool to elucidate disease etiology. Department of Epidemiology, Biostatistics & Occupational Health, McGill University, Montréal, October 2006.
135. Siemiatycki J. Keynote Speaker. Environmental causes of cancer. 28th Annual Meeting of the International Association of Cancer Registries, Goiania, Brazil, November 2006.
136. Parent M.-E, Rousseau M.-C, Siemiatycki J, Boffetta P, Cohen A. Using the workplace as a window to study the role of diesen and gasoline engine emissions in lung cancer developpement. Invited abstract submitted to the Eleventh International Congress of Toxicology, Montréal, Quebec, July 2007.
137. Siemiatycki J. Keynote Speaker. The future of occupational epidemiology? 19th International Conference on Epidemiology in Occupational Health (EPICOH 2007), Banff, October 2007. Occup. Environ. Med. 2007 Dec; 64:46.

138. Siemiatycki J. Relationship between environmental risks and health of seniors. Workshop on Seniors' Health and the environment. Health Canada, Ottawa, February 2008.
139. Siemiatycki J. Freedom of research - is it threatening or threatened? Conference of Institutional Review Boards of Quebec, (4e Journées d'étude des CER), City of Québec, Quebec, October 2008.
140. Siemiatycki J. Cancer and Environment – Annual University of Montréal Medical Faculty Assembly, Montréal, December 2008.
141. Siemiatycki J. Impact de l'environnement et du milieu de travail sur les risques de cancer : méthodologie de recherche et résultats. Conférence en santé publique, Université Laval, May 2009.
142. Siemiatycki J. CIHR and Epidemiologic Research. CSEB, Ottawa, May 2009.
143. Siemiatycki J. Mode de vie, milieu de vie: les causes modifiables du cancer. (Lifestyles and environment: modifiable causes of cancer). Keynote address. Conference nationale pour vaincre le cancer, Montréal April 2010.
144. Siemiatycki J. Montréal case-control studies on occupation and cancer. Presentation for II International Course on occupational cancer. Instituto Nacional de Cancerologia, Bogota, Colombia, August 2010.
145. Siemiatycki J. Modifiable causes of cancer and estimates of attributable fractions. Presentation for II International Course on occupational cancer, Instituto Nacional de Cancerologia, Bogota, Colombia, August 2010.
146. Siemiatycki J. Asbestos and cancer in Quebec: a presentation of studies in three populations. Presentation for II International Course on occupational cancer. Instituto Nacional de Cancerologia, Bogota, Colombia, August 2010.
147. Siemiatycki J. An overview of recognized environmental and lifestyle causes of cancer, and their contribution to the overall burden of cancer. International Congress of Pathophysiology, Montréal, September 2010.
148. Siemiatycki J. Les causes modifiables du cancer (Lifestyles and environment: modifiable causes of cancer). Conference annuelle de la Société du cancer du Canada, division Québec, November 2010.
149. Siemiatiycki J. Alison McDonald's research on the impact of Medicare in Québec. Department of Epidemiology and Biostatistics, McGill University, Montréal, Quebec, May 2011.
150. Siemiatycki J. An overview of environmental causes of cancer. Special Symposium to honour Nobel Prize winner CRCHUM, Montréal, Quebec, June 2011.
151. Siemiatycki J. Review of IARC evaluation on cellphones and cancer. Congress of Epidemiology, Montréal, Quebec, June 2011.
152. Siemiatycki J. L'évidence concernant les risques de cancer liés à l'utilisation du téléphone cellulaire. Institut national de santé publique du Québec, October 2011.
153. Siemiatycki J. Do cellphones cause brain cancer? Canadian Cancer Research Conference, Toronto, November 2011.
154. Siemiatycki J. Do cellphones cause brain cancer? Canadian Center for Architecture. Public science lecture series, Montréal, Quebec, January 2012.
155. Siemiatycki J. Do cellphones cause brain cancer? McGill University Department of Epidemiology lecture series, Montréal, Quebec, March 2012.
156. Siemiatycki J. L'environnement et le risque de cancer. Table ronde. Conference annuelle de la Coalition Cancer, Montréal, Quebec, March 2012.
157. Siemiatycki J. An Overview of Modifiable Risk Factors for Cancer. CHUM Department of Medicine, Montréal, Quebec, March 2012.
158. Siemiatycki J. Do cell phones cause brain cancer? The epidemiologic evidence. Grand Rounds, St-Mary's Hospital, Montréal, Quebec, September 2012.
159. Siemiatycki J. The epidemiology of cell phones and brain cancer. Centre hospitalier universitaire Vaudois, Lausanne, Suisse, October 2012
160. Siemiatycki J. Occupational causes of cancer. Annual meeting of Occupational & Environmental Medical Association of Canada, Montréal, Quebec, September 2013.
161. Siemiatycki J. Fraction of lung cancer that is legally attributable to smoking: a novel parameter. ISPED, Bordeaux, France, November 2013.

162. Siemiatycki J. Some challenges in environmental cancer research. Boston University School of Public Health, Boston, Massachusetts, February 2014.
163. Siemiatycki J. Les causes modifiables du cancer: le cancer peut être évité. Symposium de La Fondation Sauve Ta Peau, Montréal, Quebec, September 2014.
164. Siemiatycki J. Using epidemiologic research to combat the tobacco industry. BIPS, Bremen, Germany, September 2015.
165. Siemiatycki J. Insights into the use of epidemiologic data in a class action lawsuit against the tobacco industry. CRCHUM division seminar, Montréal, Quebec, September 2015.
166. Siemiatycki J. Development of a methodology to estimate legally attributable fraction of lung cancer attributable to cigarette smoking. McGill Univ Dept of Epidemiology, Montréal, Quebec, October 2015.
167. Siemiatycki J. Using epidemiologic research to combat the tobacco industry. SIRIC-BRIO Cancer Centre. Bordeaux, France, November 2015.
168. Siemiatycki J. Do cell phones cause brain cancer? The epidemiologic evidence. Dept of Medicine, CHUM, Montréal, Québec, November 2015.
169. Siemiatycki J. Occupation and cancer. Conference for the 50th Anniversary of IARC, Lyon, June 2016.
170. Siemiatycki J. Contribution of epidemiology to knowledge on occupational risk factors for cancer. 34e Congrès national de Médecine et Santé au Travail, Paris, France, June 2016.
171. Siemiatycki J. The influence of JC McDonald on the evolution of epidemiology in Canada. Symposium in honour of JC McDonald. McGill Univ., Montréal, Quebec, May 2017.
172. Siemiatycki J. A survey of knowledge on occupational causes of cancer. Keynote address. International Association of Cancer Registries, Utrecht, Netherlands, October 2017.
173. Siemiatycki J. La preuve statistique au tribunal : recours collectif en situation d'incertitude. Café-statistique de la Société des statisticiens français de la région parisienne, Paris, France, May 2018.

SCIENTIFIC PRESENTATIONS - OFFERED AND ACCEPTED

1. Siemiatycki J. Comparison of mail, telephone and home interview methods for health surveys. International Epidemiologic Association Meeting. Puerto Rico. August 1977.
2. Siemiatycki J, Day NE, Fabry J, Cooper, JA. Identification d'agents cancérigènes dans le milieu de travail: un nouveau système épidémiologique de monitoring. Deuxième conférence internationale sur la science des systèmes dans le domaine de la santé. Montréal, July 1980.
3. Siemiatycki J, Richardson L, Pless B. Equality in Medical Care under National Health Insurance in Montréal. Deuxième conférence internationale sur la science des systèmes dans le domaine de la santé. Montréal, July 1980.
4. Siemiatycki J. Discovering occupational carcinogens. International Symposium on Chemical Mutagenisis, Human Population Monitoring and Genetic Risk Assessment. Ottawa. October 1980.
5. Siemiatycki J, Richardson L, Gerin M. Discovering occupational carcinogens by a substance-based case-control approach-fieldwork considerations. International Epidemiologic Association Meeting. Edinburgh. August 1981.
6. Siemiatycki J, Colle E, West R, Belmonte M. Space-time clustering of juvenile-onset diabetes in Montréal. International Epidemiologic Association Meeting. Edinburgh. August 1981.
7. Siemiatycki J, Gerin M, Richardson L. Discovering occupational carcinogens by an exposure-based case-control approach: exposure assessment aspects. Second International Symposium on Epidemiology in Occupational Health. Montréal, August 1982.
8. Siemiatycki J, Gerin M, Lakhani R, Dewar R, Pellerin J, Richardson L. Nickel and ancer associations from a multicancer occupation exposure case-referent study. Symposium on Nickel in the Environment. Lyon, March 1983.
9. Gerin M, Siemiatycki J. La traduction des histoires professionnelles en histoires d'expositions chimiques: un défi pour l'hygiéniste du travail. Congrès de l'Association pour l'hygiène industrielle du Québec. Quebec, May 1983.
10. Siemiatycki J, Colle E, Campbell S, Belmonte M. Preliminary analysis of a case-control study of Type I diabetes mellitus. Baltimore, June 1985.

11. Siemiatycki J, Richardson L, Gerin M, Goldberg M, Dewar R. Associations between nine sites of cancer and nine organic dusts: results from a hypothesis-generating case-control study in Montréal. Society for Epidemiologic Research. Chapel Hill, North Carolina, June 1985.
12. Richardson L, Siemiatycki J, Gerin M, Goldberg, M, Dewar R, Desy M, Campbell S, Wacholder S. Associations between several sites of cancer and nine organic dusts: results from a case-control study. Fourth International Symposium on Epidemiology in Occupational Health. Como, Italy, September 1985.
13. Richardson L, Siemiatycki J. Case-control study methods: when to interview subjects and non-response bias. Fourth International Symposium on Epidemiology in Occupational Health. Como, Italy, September 1985.
14. Soskolne C, Jhangri G, Checkoway, Risch H, Siemiatycki J, et al. Sulphuric acid exposure in laryngeal cancer: induction and latency estimates from a lagged exposure window analysis. XII Scientific Meeting of the International Epidemiology Assoc. Los Angeles, August, 1990.
15. Payment P, Richardson L, Edwardes M, Franco E, Siemiatycki J. (1989). Gastrointestinal illness and drinking water: a prospective epidemiological study. 57th Conjoint Meeting on Infectious Diseases (CACMID), Montréal, 25-29 November 1989, Résumé C-30.
16. Payment P, Richardson L, Edwardes M, Franco E, Siemiatycki J. (1990). Drinking water related gastrointestinal illnesses. 1990 Annual Meeting of the American Society for Microbiology, Anaheim California, 13-17 May 1990.
17. Payment P, Richardson L, Edwardes M, Franco E, Siemiatycki J. (1990). A prospective epidemiological study of drinking water related gastrointestinal illnesses. International Association on water Pollution Research and Control, Health Related Water Microbiology Group, Tubingen, West Germany, 1-6 April 1990.
18. Case BA, Dufresne A, Siemiatycki J, Fraser R. Decoding occupational history from total lung particulate analysis. II: A comparative study. Brit. Occ. Hyg. Soc.; Seventh International Symposium on Inhaled Particles, Edinburgh, September 1991, S4.5.
19. Suarez-Almazor M, Soskolne C, Fung K, Jhangri G, Burch D, Howe G, Miller A, Siemiatycki J, Lakhani R, Dewar R. Choice of summary worklife exposure measures in the estimation of risk: an empirical assessment. Canadian Epidemiology Symposium. Edmonton. May. 1991.
20. Siemiatycki J, Nadon L, Dewar R. Cancer risks due to occupational exposure to polycyclic aromatic hydrocarbons. 8th International Symposium on Epidemiology in Occupational Health, Paris, France, September 1991.
21. Bourbonnais R, Siemiatycki J. Socioeconomic variables and cancer risk. Canadian Society for Epidemiology and Biostatistics. Edmonton, May 1991.
22. Gerin M, Begin D, Siemiatycki J, Dewar R. Study on the validity of the NOES job-exposure matrix using industrial hygiene measurements obtained in Montréal. Conference on Retrospective Assessment of Occupational Exposure. IARC Lyon. April 1994.
23. *Camus M, Siemiatycki J. Estimating past asbestos fiber levels in the general population of asbestos mining towns in Quebec. International Society Environmental Epidemiology, Research Triangle Park, N.C. Sept. 1994.
24. Goldberg MS, Dewar R, Siemiatycki J. Confounding and other design issues in cancer incidence studies of hazardous waste sites. Canadian Society for Epidemiology and Biostatistics. St-John's, Newfoundland, Aug 1995.
25. Goldberg MS, Dewar R, Siemiatycki J. Confounding and other design issues in cancer incidence studies of hazardous waste sites. International Society for Environmental Epideemiology. Noordwijkerhout, Netherlands, Aug, 1995.
26. Case BW, Camus M, Siemiatycki J. Trends in Pathologic Diagnosis of Malignant Mesothelioma among Quebec Women 1970-1990. Royal College of Medicine. Montréal. Sept. 1995.
27. Aronson KJ, Siemiatycki J. Dewar R, Gerin M. Occupational Risk Factors for Prostate Cancer. Canadian Society for Epidemiology and Biostatistics, St-John's, Newfoundland, Aug 1995.
28. *Camus M, Siemiatycki J. The Estimation of Past Asbestos Fiber Levels in Quebec Asbestos Mining Towns from 1900 to 1984. Canadian Society for Epidemiology & Biostatistics, St-John's, Newfoundland, Aug 1995.

29. *Camus M, Siemiatycki J, Dewar R. Non-Occupational Asbestos Exposure and Risk of lung Cancer in the Female Population of Asbestos-Mining Towns: Implications for Risk Assessments. Canadian Society for Epidemiology and Biostatistics Meeting, St-John's, Newfoundland, Aug 1995.
30. Payment P, Franco E, Siemiatycki J, Richardson L, Renaud G, Prevost M. Epidemiology studies of tap-water related gastrointestinal illnesses. Water Quality Technology Conference, New Orleans, Nov. 1995.
31. *Fritschi L, Siemiatycki J. Self-assessed versus expert-assessed occupational exposures. Canadian Society for Epidemiology and Biostatistics Meeting, St Johns, Newfoundland, Aug 1995.
32. Payment P, Siemiatycki J, Richardson L, Renaud G. Épidémiologie des maladies gastro-intestinales et respiratoires: incidence, fraction attribuable à l'eau et coûts pour la société. ACFAS, Montréal, May 1996.
33. *Fritschi L, Parent M-É, Siemiatycki J. Gastric cancer and occupation. Australasian Epidemiological Association, Victoria, Australia. July 1996.
34. *Camus M, Case BW, Siemiatycki J. Risk assessment for women living in chrysotile mining towns.1: Environmental exposure assessment. Fourth International Mesothelioma Conference, Philadelphia, May 1997.
35. Case BW, Camus M, Siemiatycki J. Risk assessment for women living in chrysotile mining towns.2: Mesothelioma: observed vs. predicted. Fourth International Mesothelioma Conference, Philadelphia, May 1997.
36. *Camus M, Siemiatycki J. Cancer risks due to non-occupational asbestos exposure. Can. Soc. for Epidemiol. & Biostat. London, Ontario, May 1997.
37. Weston TL, Aronson KJ, Howe GR, Nadon L, Siemiatycki J. Cancer mortality risk in a cohort of working men. Canadian Society for Epidemiology and Biostatistics, London, Ontario, May 1997.
38. *Parent M-É, Siemiatycki J, Menzies L, Fritschi L, Colle E. Can Bacille-Calmette Guérin vaccination prevent insulin-dependent diabetes mellitus (IDDM)? Canadian Society for Epidemiology and Biostatistics, London, Ontario, May 1997.
39. Wolf, S, Siemiatycki J, Beyersmann, D, Jockel, K. H. A case-control study of lung cancer - performance of a job-exposure matrix for cadmium, chromium, nickel, and stainless steel dust. Internat. Epidemiol. Assoc. European Region Meeting. Munster, Germany, Sept. 1997.
40. *Parent, M.E. Siemiatycki J. Exposition professionnelle aux émissions d'essence et de diesel, et cancer du poumon. ACFAS, Quebec, May 1998.
41. *Parent M-É, Siemiatycki J, Boffetta P. Occupational exposure to gasoline and diesel engine emissions and lung cancer. Soc. Epid. Res, Chicago, June 1998.
42. *Parent M-É, Siemiatycki J, Boffetta P, Cohen A. Occupational exposure to gasoline and diesel exhausts and lung cancer. Inter. Soc. Environ. Epid, Boston, August 1998.
43. *Parent M-É, Siemiatycki J, Boffetta P, Cohen A. Gasoline and diesel engine emissions in the workplace and lung cancer. PREMUS-ISEOH '98, Helsinki, Finland, Sept. 1998.
44. Leffondre K, Abrahamowicz M, Rachet B, Siemiatycki J. Modeling smoking history: A comparison of different approaches. Congress of Epidemiology, Toronto, June 2001.
45. Fritschi L, Nadon L, Benke G, Lakhani R, Latreille B, Parent M-É, Siemiatycki J. Validation of expert assessment of occupational exposures X2001 – Occupational Exposure Assessment for Epidemiology and Practice, Gothenburg, Sweden, June 2001.
46. Parent M-É, Siemiatycki J, Desy M. Case-control study of occupational exposures and risk of prostate cancer among farmers. Case-control study of occupational exposures and risk of prostate cancer among farmers, Toronto, June 2001.
47. Siemiatycki J, Camus M, Parent M-É, Richardson L, Desy M, Case BW. Case-control study of pleural mesothelioma among women in Quebec chrysotile mining regions. Inhaled Particles IX (BOHS), Cambridge, United Kingdom, September 2001.
48. Jockel K-H, Wolf S, Ahrens W, Jahn I, Pohlabeln H, Beyersmann D, Siemiatycki J. Cadmium as a human lung carcinogen. Jahrestagung der Deutschen Arbeitsgemeinschaft fur Epidemiologie (DAE) [Annual convention of the German epidemiology working group], Garmisch-Partenkirchen, Germany, September 2001.

49. Leffondre K, Abrahamowicz M, Siemiatycki J. Comparison of Cox's model versus logistic regression for case-control data with time-varying exposure: a simulation study. Annual Meeting of the Statistical Society of Canada (SSC). Hamilton, Ontario. May 2002.
50. Parent M-É, Siemiatycki J, Desy M. Association between Alcohol Consumption and Each of 23 Types of Cancer in Men. Soc. Epid. Res, Palm Desert, California, June 2002.
51. Leffondre K, Abrahamowicz M, Siemiatycki J. Comparison of Cox's model versus logistic regression for case-control data with time-varying exposure: a simulation study. Society for Epidemiologic Research (SER), Palm Desert, California, June 2002.
52. Rachet B, Parent M-É, Siemiatycki J. Welding Fumes and Lung Cancer: A Case-Control Study, Soc. Epidemiol. Res, Palm Desert, California, June 2002.
53. Rachet B, Abrahamowicz M, Sasco A, Siemiatycki J. Flexible estimation of the distribution of lag in the effects of exposures and interventions. 34th Annual SER Meeting. Palm Desert, California. June 2002.
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161. *Xu M, Richardson L, Campbell S, Pintos J, Siemiatycki J. Time trends and study design determinants of response rates in case-control studies of cancer. 18th Congress of Students, Interns and Residents of CRCHUM, Montréal, Quebec, 4 May 2016.
162. *Karumanchi S, Hatsopoulou M, Richardson L, Thierry B, Goldberg M, Siemiatycki J. Land use regression model of UFPs in the Grand Montréal Region. Oral presentation. Canadian Society for Epidemiology and Biostatistics, Winnipeg, Manitoba, 8-10 June 2016.
163. *Xu M, Richardson L, Campbell S, Pintos J, Siemiatycki J. Subject response rates in case-control studies of cancer: quality of reporting, time trends, and study design determinants. Epidemiology Congress of the Americas, Miami, Florida, 21-24 June 2016.
164. *Rémen T, Siemiatycki J, Lavoué J. Impact of inter-coder differences in occupation and industry classification coding on exposure estimates obtained via job-exposure matrix: example of gasoline engine

emissions in CANJEM. Oral presentation. 25th EPICOH - Epidemiology in Occupation Health Conference, Barcelona, Spain, 4-7 September 2016.

165. *Sauvé JF, Lavoué J, Siemiatycki J, Parent ME. Evaluation of a hybrid expert approach for retrospective assessment of occupational exposures in a population-based study of prostate cancer in Montréal, Canada. Oral presentation. 25th EPICOH - Epidemiology in Occupation Health Conference, Barcelona, Spain, 4-7 September 2016.

166. *Pasquet R, Cardis E, Richardson L, Lavoué J, Siemiatycki J, Koushik A. The association between occupational exposure to metals and metalloids and brain cancer risk. 25th EPICOH - Epidemiology in Occupation Health Conference, Barcelona, Spain, 4-7 September 2016.

167. Russ D, Rémen T, Ho KY, Chow WH, Davis F, Hofmann J, Huang H, Purdue M, Schwartz K, Siemiatycki J, Zhang Y, Silverman D, Johnson C, Lavoué J, Friesen M. Recommendations for prioritizing expert review of free-text job descriptions that underwent computer-based coding using the SOCcer algorithm. 25th EPICOH - Epidemiology in Occupation Health Conference, Barcelona, Spain, 4-7 September 2016.

168. *Sauvé JF, Labrèche F, Richardson L, Goldberg MS, Parent MÉ, Siemiatycki J, Lavoué J. Development of the CANJEM Canadian general-population job-exposure matrix from past expert evaluations. Oral presentation. Canadian Association for Research on Work and Health (CARWH) conference, Toronto, Ontario, October 2016.

169. *Rémen T, Siemiatycki J, Lavoué J, Verner MA. Impact of inter-coder differences in occupation and industry classification coding on exposure estimates obtained via job-exposure matrix: example of gasoline engine emissions in CANJEM. Poster. International Society of Exposure Science (ISES) 2016 Annual Meeting, Utrecht, Netherlands, 9-13 October 2016.

170. Grundy A, Ho V, Parent ME, Siemiatycki J, Koushik A. Lifetime recreational moderate-to vigorous physical activity and the risk of ovarian cancer by subtype. Poster presentation. 2016 American Institute for Cancer Research (AICR) Research Conference, North Bethesda, Maryland,14-16 November 2016.

171. Grundy A, Ho V, Parent ME, Siematycki J, Koushik A. The impact of menopausal status on the association between moderate-to-vigorous physical activity among participants in the Prevention of OVArian Cancer in Quebec (PROVAQ) study. Oral Presentation. Canadian Society for Epidemiology and Biostatistics 2017 Biennal Conference, Banff, Alberta, 1 June 2017

172. Bowman JD, Vila J, Richardson L, Kincl L, Cardis E on behalf of the INTEROCC Study Group. Occupational Exposures to Radio-frequency Electric Fields Assessed for the INTEROCC Study of Brain Cancer. Oral presentation. American Industrial Hygiene Association conference, Seattle, Washington, 4-7 June 2017.

173. *Karumanchi S, Siemiatycki J, Hatzopoulou M. Some challenges in measuring ultra-fine particles and developing a land use regression model. Oral presentation. Canadian Society for Epidemiology and Biostatistics (CSEB) 2017 Biennal Conference, Banff, Alberta, 30 May 2017.

174. *Sauvé JF, Davies HW, Parent MÉ, Peters CE, Siemiatycki J, Sylvestre MP, Lavoué J. Development of quantitative estimates of wood dust exposure in a Canadian general population job-exposure matrix based on past expert assessments. 26th Conference on Epidemiology in Occupational Health (EPICOH 2017), Edinburgh, Scotland, August 2017.

175. Ho V, Xu M, Pintos J, Lavoué J, Abrahamowicz M, Rousseau M.C, Richardson L, Siemiatycki J. Occupational exposures to leaded and unleaded gasoline engine emissions and lung cancer risk. Canadian Cancer Research Conference, Vancouver, British Columbia, 5-7 November 2017.

176. Lequy E, Siemiatycki J, Leblond S, et al. Moss biomonitoring as an alternative to assess exposure to atmospheric metals in environmental epidemiology: the example of the bramm network and the gazel cohort. Poster. SEE Young 2018, Early Career Researchers Conference on Environmental Epidemiology – Together for a Healthy Environment, Freising, Germany, 19–20 March 2018. Occup Environ Med 2018;75:A27.

177. Ho V, Parent MÉ, Lavoué J, Zhu Y, Siemiatycki J, Koushik A. Gender Differences in Occupational Physical Activity. Oral presentation. ISES-ISEE 2018 Joint Annual Meeting, Ottawa, Ontario. 26-30 August 2018.

178. *Xu M, Ho V, Siemiatycki J. Association between occupational exposure to textile fibre dusts and lung cancer in a population-based case-control study in Montréal: a preliminary analysis comparing results from three analytical methods. Oral presentation. ISES-ISEE 2018 Joint Annual Meeting, Ottawa, Ontario, 26-30 August 2018.
179. Zhu Y, Lavoué J, Parent MÉ, Siemiatcyki J, Koushik A, Ho V. Occupational Physical Activity and Lung Cancer Risk among Participants of the Alberta's Tomorrow Project. Poster. ISES-ISEE 2018 Joint Annual Meeting, Ottawa, Ontario, 26-30 August 2018.
180. *Karumanchi S, Siemiatycki J, Richardson L, Hatzopoulou M. Estimating exposure to Ultrafine Particles in the Greater Montreal Area among case-control study subjects: Comparison of classical land use regression model with a model based on Bayesian principles - Proposal. Poster. ISES-ISEE 2018 Joint Annual Meeting, Ottawa, Ontario, 26-30 August 2018.
181. van Tongeren M, Dirkx E, Lavoué J, Siemiatycki J, Ho V. Assessment of Occupational Exposure to Endocrine Disrupting Agents. Poster. ISES-ISEE 2018 Joint Annual Meeting, Ottawa, Ontario, 26-30 August 2018.

* First author was under supervision of J. Siemiatycki when this work was carried out

GRANTS AND CONTRACTS RECEIVED

1. Comparison of three methods for conducting household health surveys; Nat. Health Res & Devel. Prog. (NHRDP); $27,000; 1974-76.
2. Pilot study of a case-control monitoring system for discovering occupational carcinogens; Conseil de la recherche en santé (CRSQ); $80,000; 1978-1980.
3. Établissement du jeune chercheur; CRSQ; $15,000; 1979-80.
4. Analyse de santé auprès de 1600 ménages montréalais; Ministère des affaires sociales (MAS); $12,708; 1980
5. Dépistage des facteurs cancérigènes de l'environnement professionnel montréalais: étude pilote; Commission des accidents du travail; $59,093 ; 1980-82.
6. Registry of patients with Juvenile Onset Diabetes in Québec; NHRDP; $35,478*; 1980-85; (P.I. Dr E. Colle).
7. Secondary analysis of a health survey in Montréal: methodologic issues and comparison of morbidity and health care utilization between social groups; NHRDP-H&W Can.; $15,000; 1981-82.
8. Exposure-based case-control approach to discovering occupational carcinogens; NHRDP-H&W Can.; $129,258; 1981-83.
9. An exposure-based case-control approach to discovering occupational carcinogens; NCIC; $131,842; 1981-83.
10. Variation in sex ratios of cancer between geographic areas; NCIC; $3,227; 1982-84.
11. Équipe associée en épidémiologie des cancers professionnels (Team grant); Institut de la recherche en santé et sécurité du travail (IRSST); $1 120,000; 1982-85.
12. Formaldehyde et cancer; IRSST; $9,500; 1983.
13. Retrospective cohort study in the Montréal fur industry; IRSST; $34,019; 1983-85.
14. Statistical analysis of a case-control study designed to discover occupational carcinogens; NHRDP-H&W Can.; $484,022; 1985-87.
15. Completion of chemical coding of exposures in a case-control study designed to discover occupational carcinogens; IRSST; $102,180; 1986.
16. Risks of cancer due to exposure to asbestos in a range of occupations; IRDA; $61,206; 1986-87.
17. Biological estimation of exposure: a tissue registry for the identification and quantification of occupational carcinogens; NCIC; $3,500*; 1986-87; (P.I. Dr B. Case)
18. Development of a proposal to study cancer risk and non-occupational exposure to asbestos; H&W Can.; $29,500; 1987-88.
19. Evaluation of cancer risk and occupational exposure to formaldehyde; H&W Can.; $30,000; 1987-88.
20. A genetic-epidemiologic study of breast cancer; NIH-NCI; $90,945(US)*; 1987-92; (P.I. Dr. R. Haile).

21. Scholar award; NHRDP-H&W Can.; $298,689; 1987-93.
22. An intervention trial to assess the risks of gastro-intestinal illness associated with consumption of treated tap water; NHRDP; $225,000*; 1987-89; (P.I. Dr P. Payment).
23. Evaluation of cancer risk and occupational exposure to polycyclic aromatic hydrocarbons; H&W Can.; $29,500; 1988-89.
24. Evaluation of cancer risk and occupational exposure to benzene, toluene and xylene; H&W Can, $40,000; 1988-89.
25. Health risks due to chrysotile asbestos in the non-occupational environment: a workshop to evaluate a research protocol; H&W Can, $20,000; 1988-89.
26. A population-based, case-control study of occupational exposure to sulphuric acid and the development of laryngeal cancer: an augmented secondary data analysis; NHRDP; $11,120*; 1988-89; (P.I. Dr. C. Soskolne).
27. Mortality due to asbestos in the general environment of the Quebec mining areas; H&W Can.; $130,000; 1989-90.
28. A case-control approach to discovering occupational carcinogens: an analysis of data; NHRDP; $55,508; 1989-90.
29. Continued analysis of a large case control study of many types of cancer: occupational and non-occupational risk factors; NHRDP; $463,827 1988-1992
30. Risk of cancer due to cigarette smoking - results of a multi-site case-control study; H&W Can.; $30,000; 1989-90.
31. Étude sur la validité de matrice emploi-expositions multisectorielles; IRSST; $18,207*; 1990-1992; (P.I. Dr. M. Gérin).
32. Équipe en épidémiologie environnementale (Team grant in environmental epidemiology) ; Fonds de recherche en santé du Québec; $526,297; 1990-1994.
33. Leukemia in children due to parental occupational exposures; NHRDP; $108,000*; 1990-1994; (P.I. Dr Claire Infante-Rivard).
34. Risk of cancer due to exposure to chlorinated solvents - results of a multi-site case-control study; H & W Can.; $30,000; 1991-92.
35. Non-occupational exposure to Quebec chrysotile asbestos and risk of cancer retrospective assessment of exposure; H & W Can.; $60,000; 1991-92.
36. Feasibility of epidemiologic methods to investigate health outcomes near waste sites; H & W Canada; $33,000; 1991-92
37. A pilot study to evaluate the prevalence of hip arthritis in the Montréal urban setting, and an evaluation of methods of recruitment of a population aged 65+; Montréal General Hospital Clinical Epidemiology; $15,000*; 1991-92; (P.I. Dr. J. Esdaile).
38. Non-occupational exposure to Quebec chrysotile asbestos and risk of cancer; mesothelioma ascertainment; NHRDP; $164,000; 1991-95.
39. Multivariate Regression Analyses of Occupational Risk Factors for Several Types of Cancers; NHRDP; $128,827; 1992-96.
40. Development of a Job-Exposure Matrix for Use in Epidemiologic Case-Control Studies of Occupational Risk Factors; NHRDP; $85,003; 1992-95.
41. A prospective epidemiological study of gastrointestinal health effects due to consumption of drinking water. E.P.A. (US)/ NHRDP/ Nat. Water Res. Inst.; $300,000*; 1993-95. (P.I.: Dr. P. Payment)
42. A population-based, case-control study of occupational exposure to acidifying agents and the development of lung cancer: an augmented, secondary data analysis. NHRDP; $72,220*; 1993-1995. (P.I. Dr. C. Soskolne).
43. Scholar award; NHRDP-Health Canada; $126,990; 1993-95.
44. Équipe en épidemiologie environnementale (Team grant in environmental epidemiology) ; Fonds de recherche en santé du Québec; $242,652; 1994-1998.
45. Examen pathologique de cas présumés de mésothéliome recensés chez des femmes depuis 1970 dans des hôpitaux du québec. Health and Welfare Canada. $30,000. 1994.

46. Cohort Study of a Ten Percent Sample of the Canadian Labour Force. NHRDP; $12,000*; 1994-97. (P.I. Dr. K. Aronson)
47. A health survey of persons living near the Miron Quarry Sanitary Landfill site, Montréal: a pilot study. NHRDP; $88,931 ; 1994-95. (P.I. Dr. M. Goldberg)
48. Occurrence of pathogenic microorganisms in water from St Laurent hydrological basin. FRSQ/ NHRDP & St Laurent Vision 2000; 1995-97. (P.I. P Payment)
49. Case-control study of lung cancer and environmental tobacco smoke; Health Canada; $544,344; 1995-1997.
50. Case-control study of lung cancer and occupational exposures: NHRDP; $840,000.; 1995–1998.
51. Occupational exposure to solvents and risk of breast cancer; National Cancer Institute of Canada; $300,000*; 1995-1997. (P.I.: M Goldberg).
52. Scholar Award; NHRDP-Health Canada; $263,329, 1995-1998.
53. Reanalysis of US data relating general mortality to air pollution; Health Effects Institute; 1998-2000 (P.I. D Krewski)
54. A case-control study of occupational risk factors for lung cancer; Medical Research Council of Canada; $554,757, 1998-2001
55. Évaluation du risque de cancer du poumon et de mésothéliome associé à l'exposition à l'amiante chez les travailleurs de la région montréalaise; Ministère de la Santé et des Services sociaux; $12,000. 1998.
56. Feasibility of a case-control study of the association between cell phone use and brain, salivary gland cancer and acoustic neurinoma. International Agency for Research on Cancer; $12,000, 1998.
57. Inorganic particulate retained dose markers in lung cancer and mesothelioma. CIHR (P.I. Bruce Case) $66,096. 1999-2003
58. Distinguished Scientist Award, Medical Research Council of Canada; $330,000; 1999-2004.
59. Évaluation du risque de mésothéliome associé à l'exposition à l'amiante chez les femmes de la région minière; Ministère de la Santé et des Services sociaux; $27,500. 1999-2000.
60. Program of research in environmental epidemiology of cancer (a national program to enhance capacity to conduct research) PREECAN; National Cancer Inst of Canada; $1,000,000; 2000-2004.
61. Designing a national research agenda in environmental epidemiology of cancer. Medical Research Council of Canada Opportunities Program; $40,000; 2000-2001.
62. Multi-centric case-control study of cell phone use and cancer risk in Montréal. CIHR; $500,000; 2000-2004.
63. Trainee award for: Bernard Rachet, Post-doctoral fellow. PREECAN – NCIC; $46,750; 2001-2003.
64. Cardiogene: a consortium to explore the gene-environment paradigm of major cardiovascular disorders in human and animal models. Canadian Institutes of Health Research, (P.I. P. Hamet) $2,632,272; 2001-2007.
65. Canada Research Chair in Environmental Epidemiology. Federal CRC program. $1,400,000; 2001-2008.
66. Installation of CRC. Canadian Foundation for Innovation. $312,000; 2002-2004.
67. Occupational and lifestyle factors in the etiology of prostate cancer, and establishing a platform for studying susceptibility biomarkers (Part 1). Canadian Cancer Society, Prostate Cancer Research Initiative, National Cancer Institute of Canada, (P.I. M-É Parent) $947,360; 2002-2007.
68. Center for research on environmental etiology of cancer. For the application process. Centre Hospitalier de l'Université de Montréal (CHUM); $7,000; 2002-2003.
69. Traffic-related air pollution and socioeconomic gradients in the incidence of cancer. CIHR, (P.I. M Goldberg) $497,000; 2004-2007.
70. Development and validation of new statistical methods for modeling intermediate events in survival analysis. CIHR, (P.I. M Abrahamowicz) $68,250; 2004-2005.
71. New survival analytic methods for time-dependent exposures in case-control studies, with applications to cancer. CIHR (P.I. K Leffondré) $52,791; 2004-2007.
72. Trainee award for: Venkata Ramana Kumar, Post-doctoral fellow. PREECAN – NCIC; $66,000; 2004-2007.

73. Environmental Cancer Research Team. Development grant for the preparation of the full team grant application. CIHR (P.I. J. Siemiatycki) $9,500; 2005-2006.
74. Trainee award for: Franco Momoli, PhD student. PREECAN – NCIC; $25,600; 2005-2006.
75. Occupational and selected non-occupational risk factors for lung cancer: Analysis of a case-control study in Montréal. CIHR (co-P.I.'s: J Siemiatycki & M-É Parent) $1,920,447; 1999.2011.
76. Development and evaluation of a cost-effective approach for retrospective assessment of occupational exposures in population-based studies (pilot study). Canadian Cancer Etiology Research Network - NCIC (P.I. M-É Parent) $35,000; 2006-2007.
77. Trainee award for: Aihua Liu, PhD student. PREECAN – NCIC; $12,600; 2006-2007.
78. Prostate cancer and occupational whole body vibration. Ontario Workplace Insurance Board: Research Advisory Council; Solutions for Workplace Change (P.I. J Purdham); $140,480; 2006-2008.
79. Guzzo-SRC Chair in Environment and Cancer. Cancer Research Society, $1,285,000; 2007-2020.
80. INTEROCC: Occupational exposures and brain cancer. NIH (P.I. E Cardis: To support the analysis of the occupational component of an international case-control study involving 13 countries and coordinated at the International Agency for Research on Cancer of the WHO [France]); $1,626,757 US; 2008-2010.
81. Development and validation of a lung cancer risk prediction model. NCIC (P.I. I Karp); $102,099; 2008-2010.
82. Occupational and lifestyle factors in the etiology of prostate cancer, and establishing a platform for studying susceptibility biomarkers (Part 2). NCIC (P.I.: M-É Parent); $756,000; 2008-2011.
83. Preparation and development of an epidemiological study of modifiable and genetic factors associated with ovarian cancer risk (pilot project). Ovarian Cancer Canada (P.I.: A Koushik); $28,330; 2008-2009.
84. SYNERGY - Pooled analysis of case-control studies on the joint effects of occupational carcinogens in the development of lung cancer: Montréal component. German Statutory Accident Insurance (DGUV) (P.I.: A Koushik); $119,177; 2008-2010.
85. The risk of lung cancer related to occupational and recreation physical activity and to dietary intake of flavonoids. Canadian Cancer Research Society. (P.I.: A Koushik); $208,317; 2009-2012.
86. A case-control study of modifiable and genetic factors associated with the risk of ovarian cancer. Canadian Cancer Society Research Institute (P.I: A Koushik); $498,997; 2010- 2013.
87. Occupational and selected nonoccupational risk factors for lung cancer: analysis of a case-control study in Montréal. CIHR (P.I: J Siemiatycki, M-É Parent); $850,620; 2011-2015.
88. Quebec Research Program for Prostate Cancer Prevention. Cancer Research Society (P.I.: M-É. Parent, P Karakiewics) $4,728,203; 2011-2015.
89. Extreme weather and maternal-child health: targeting future impacts of climate change. CIHR. (P.I.: N Auger) $85,333; 2015-2019.
90. Development of an instrument for assessing occupational exposures in cancer case-control studies and its application to cancers of lung, brain, ovary. Cancer Research Society- Programme GRePEC (Groupe de recherche et de prévention en environnement-cancer). (P.I.: J Siemiatycki, M Pollak) $2,510,890; 2011-2018.
91. Occupational physical activity and lung cancer. (P.I.: V Ho, A Koushik).CIHR. $75,000. 2017-2018.
92. Analyses of existing Canadian cohorts and databases related to occupational physical activity and lung cancer risk. CIHR. (P.I.: V Ho, A Koushik) $74,989; 2017-2018.
93. The role of lifestyle factors in ovarian cancer prognosis. Department of Defence – Ovarian Cancer Research Program. (P.I.: A Koushik) $216,458 USD (est. $293, 000 CAD); 2015-2017. Extended August 2018.
94. Occupational Exposure to Endocrine Disrupting Chemicals and Colorectal Cancer risk. CIHR (P.I.: V Ho, J Siemiatycki) $252,450; 2018-2021.
95. Occupational exposures of women: improvement of an existing job exposure matrix to provide gender-specific estimations of exposure. IRSST. (P.I.: V Ho) $491,484; 2018-2021.